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JANUARY
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VOL LVI
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TABLE OF CONTENTS

JANUARY, 1955

ORIGINAL ARTICLES

Six Years Experience with Dicumarol Prophylaxis against Postoperative Thrombo-Embolic Complications in Private Practice—Harry R. Custer, M.D., Colby, Kansas and Russell L. Mustard, M.D., Battle Creek, Michigan . 1

Salicylate Poisoning—Vincent Ricciutti, M.D., Buffalo, New York 5

Laparotomy Incisions—Barrett A. Nelson, M.D., Manhattan, Kansas 9

Sunlight and Cancer—Herbert L. Ketterman, Kansas City, Kansas 12

EDITORIALS

Our New Cover Page 17

Federal Aid in Kansas, Part IV 17

Help for the Afflicted 19

Medical Provisions for Tax Returns . . . 19

Tribute to a Friend 20

MISCELLANEOUS

Just Browsing 15

President's Page 16

Peripheral Embolic Arterial Occlusion—Eugene L. Petry, M.D.—Senior Thesis 30

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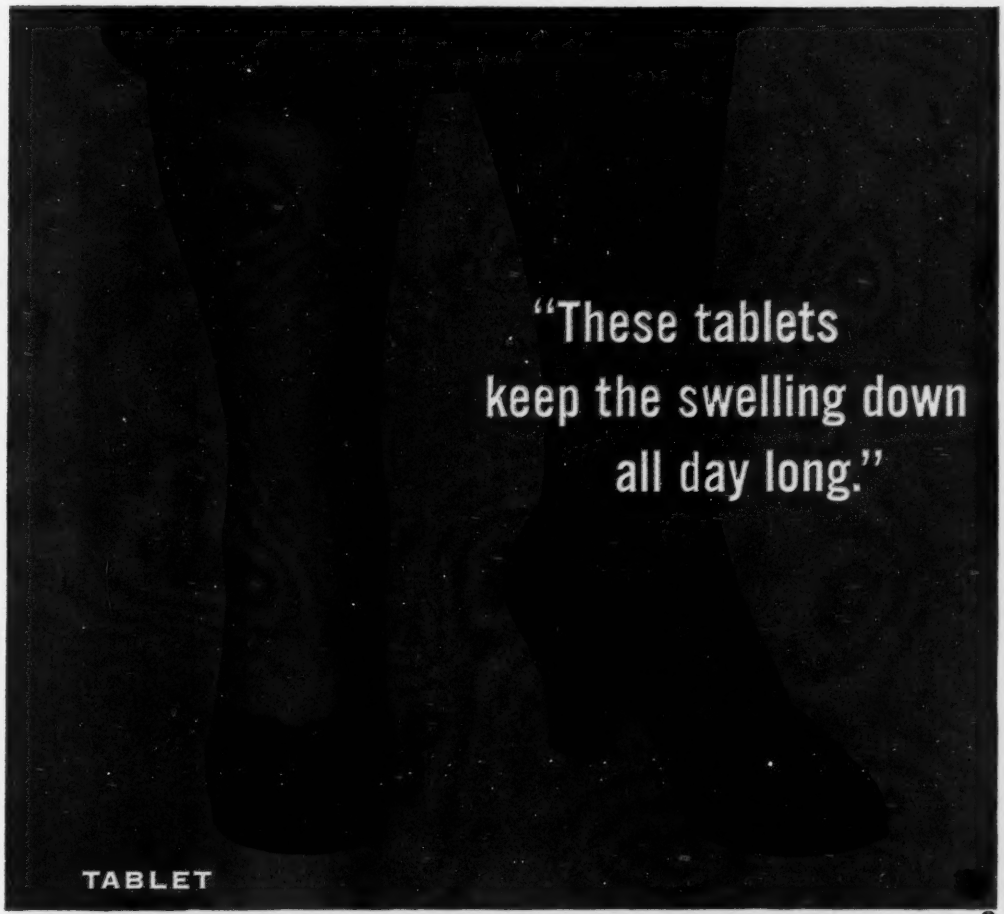

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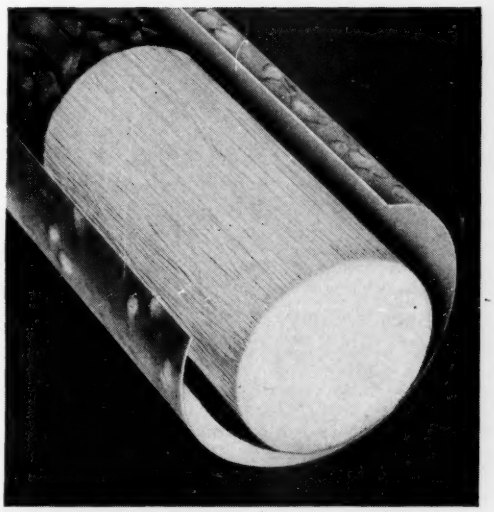

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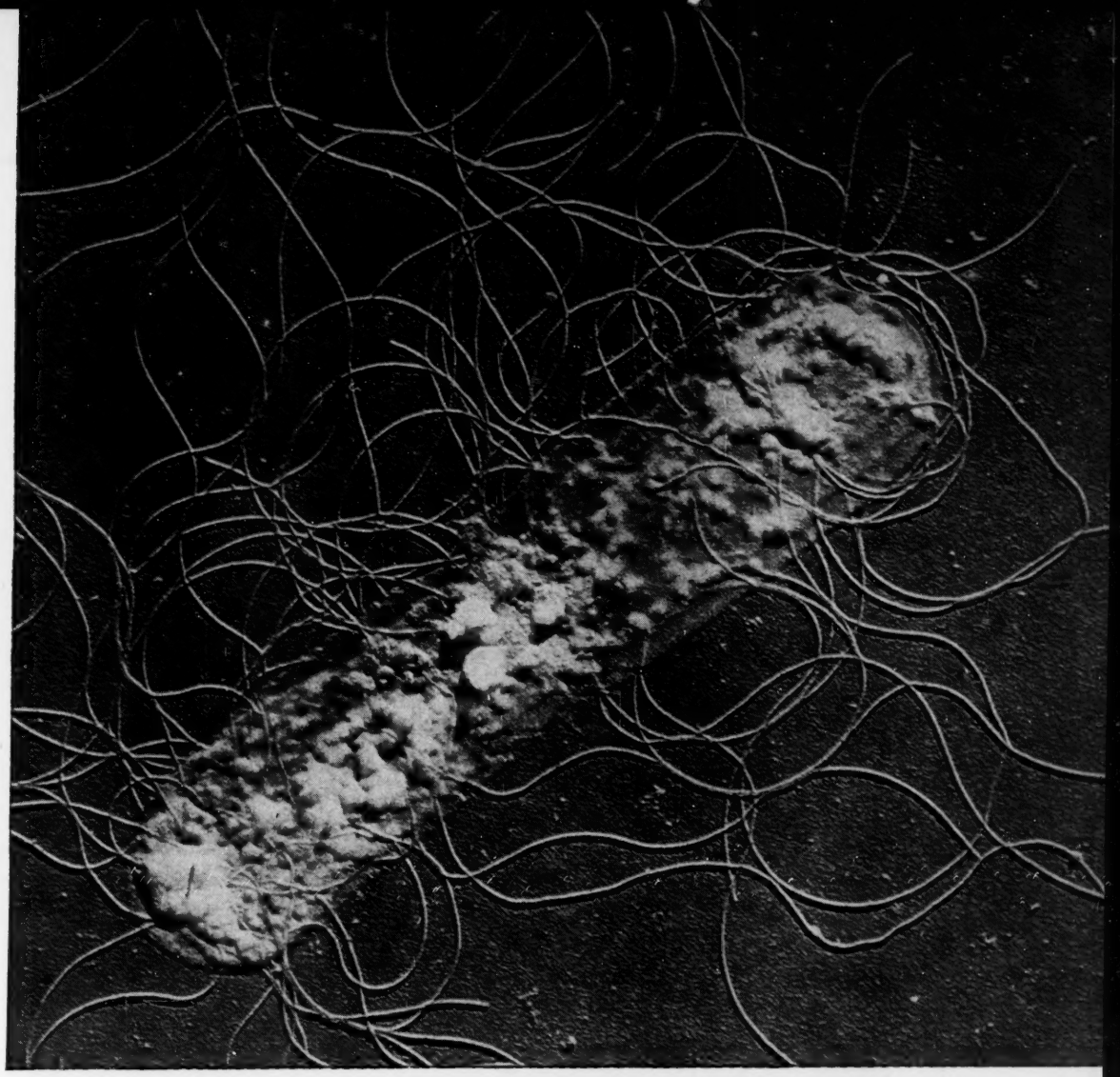

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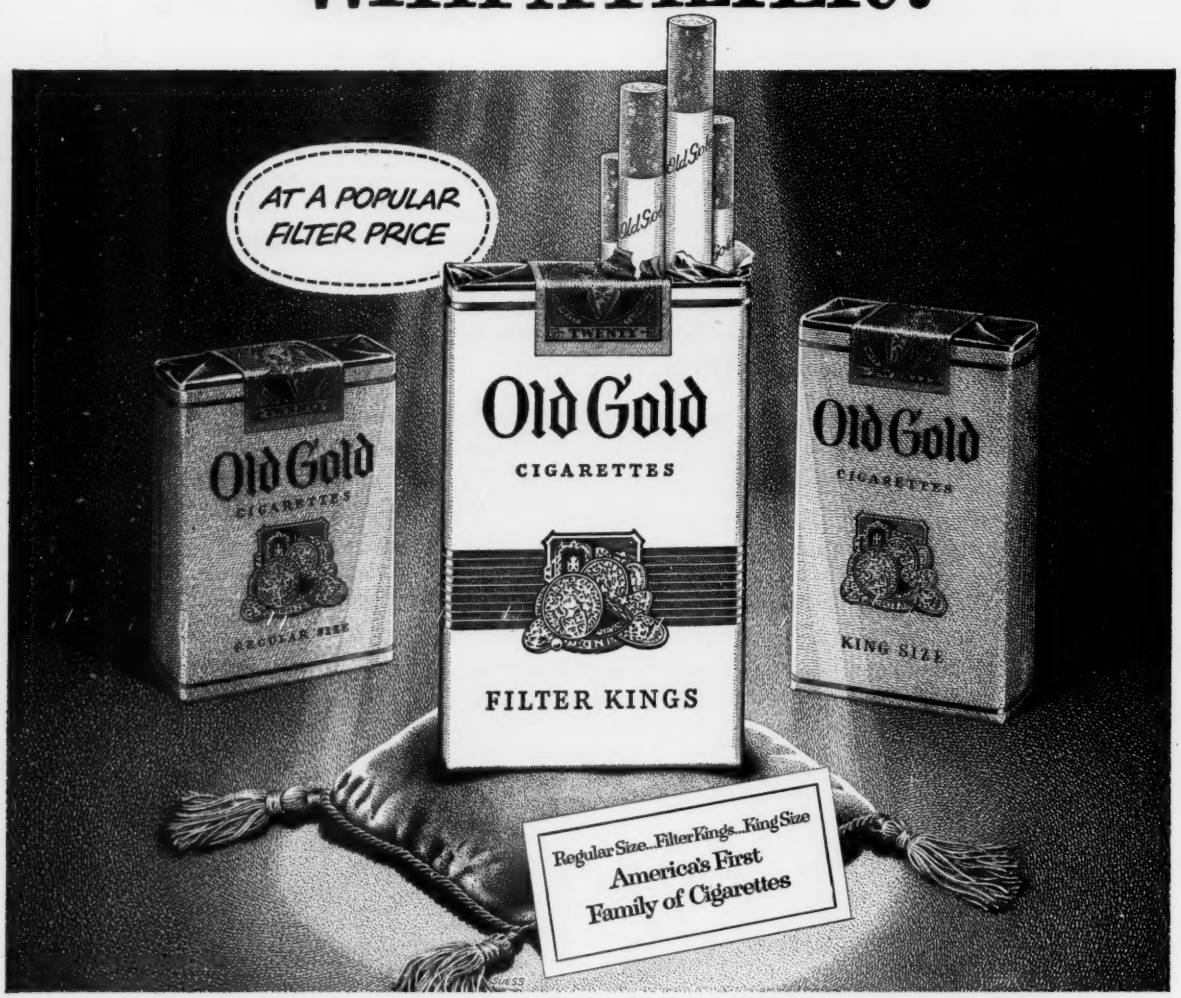

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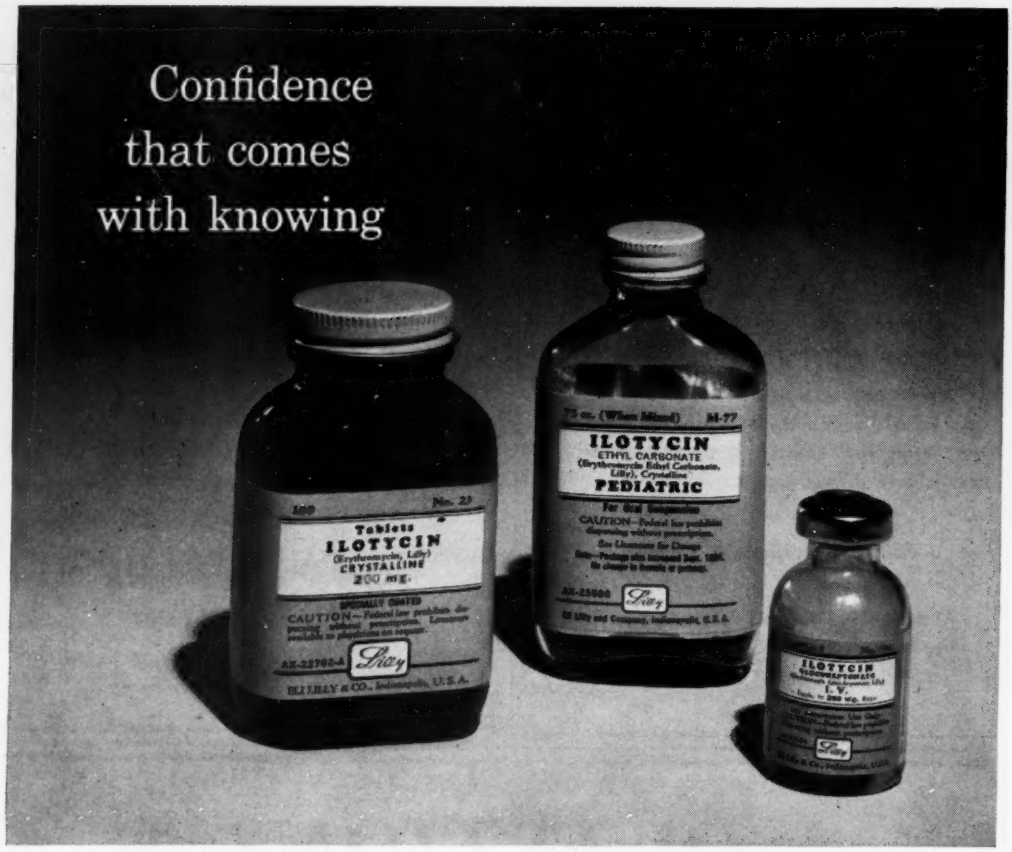

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Volume LVI JANUARY, 1955 No. 1

Six Years Experience with Dicumarol Prophylaxis against Postoperative Thromboembolic Complications in Private Practice

Harry R. Custer, M.D., and Russell L. Mustard, M.D.
Colby, Kansas Battle Creek, Michigan

The private practice of surgery differs considerably from the practice of clinical and research surgery. The surgeon in private practice is obligated to employ only proven techniques and methods of therapy. Thus, the following discussion concerns a period of six years in private surgical practice and the prophylactic use of dicumarol, after its proven value, against postoperative thromboembolic complications.

All the patients involved in this study were personally examined, operated upon, treated, and followed by the authors. In this way preoperative, surgical, and postoperative uniformity was obtained. A total of 4,383 patients were operated upon between the years 1948 and 1954; of these, 331 received dicumarol prophylactically, following surgery.

In recent years there has been a steady increase in the number, variation, and extent of surgical procedures, along with an increase in the age of the surgical patient. Proportionally, there has been an increasing opportunity for the development of postoperative thromboembolic complications. Also, it has been proved that there is an unpredictable physiological increase in the activity of the blood clotting elements following the trauma of surgery.[4] The possibility of increased use of preoperative and postoperative blood transfusions and certain antibiotics causing increased coagulability of the blood has been reported.[10] Aside from the possible causes, reports show an increased incidence in postoperative thromboembolism in the last few years.[10]

The actual incidence of minimal and subclinical postoperative thrombosis and pulmonary embolism is not known. Undoubtedly many cases are misdiagnosed and are called myocardial insufficiency, cardiac failure, or chronic shock. However, it has been shown the middle aged and elderly patients who are confined to bed have more than a 50 per cent incidence of phlebothrombosis of the deep veins of the leg.[6] The same author states that fatal pulmonary embolism was responsible for 3.13 per cent of all deaths in these patients.[6]

Pratt stated that 8 per cent of his reported postoperative deaths were due to thromboembolism.[11] According to Kirby,[7] postoperative venous thrombosis has a reported incidence of from 1 to 10 per cent with pulmonary embolism being the etiology of about 5 to 7.5 per cent of all postoperative deaths.[7, 8] McCortney[9] stated that among the postoperative fatalities, thromboembolism was present in 11.5 per cent, and 5.3 per cent of the deaths were due to pulmonary emboli. In his series operations on the uterus accounted for 25.4 per cent of all postoperative thromboemboli, and surgery for herniae ac-

counted for 22.6 per cent of all postoperative fatal pulmonary emboli.

Reports have shown that there is no warning in 70 to 80 per cent of all postoperative fatal pulmonary emboli.[7, 8] Therefore, it is obvious that prophylaxis is far superior to treatment of the complication.

It is the intent of the authors to present a safe and effective method of using coumarin (dicumarol) prophylactically against postoperative thromboembolic complications, and to evaluate the efficacy of dicumarol prophylaxis when used under a closely supervised routine in private practice.

Dicumarol was preceded by heparin in the prophylaxis of thromboembolism. But today the coumarin compounds (dicumarol, tromexan, and cyclocumeral[1]) are by far the most widely used drugs in the prevention of thromboembolism. Dicumarin, the active principle in spoiled sweet clover, was isolated and crystallized and reported in the well-known classical experiments of Link and his associates.[12]

According to Quick, the action of dicumarol is primarily on the liver by inhibiting the formation of prothrombin and fibrinogen. Thus, the anti-thrombotic effect of dicumarol results from diminution in the available prothrombin and fibrinogen necessary for clot formation. It has further been shown that it takes 24 to 72 hours for dicumarol to significantly lower the prothrombin time in most patients.

Dicumarin was first given to humans in May, 1941, by Allen, Barker and Waugh.[1] Many errors were made, and refinements were necessary before dicumarol treatment was considered safe and effective. At first dosage was regulated by prothrombin time given in actual time rather than as a per cent of normal. Lee and White, Quick, and many others devised different methods of determining the prothrombin time. There were as many proposed routines of giving dicumarol as there were reports.

Numerous accounts of bleeding and frank hemorrhages from using dicumarol therapy appeared. Allen[1] originally stated that vitamin K was clinically ineffective to reduce the prothrombin time in over-dicumarolized patients. No one seemed to agree on the optimum level of prothrombin time in postoperative patients. Allen,[1] in his first report, kept the prothrombin time between 35 and 60 seconds and began giving dicumarol on the first postoperative day. Later, Allen[2] maintained a prothrombin level between 10 and 30 per cent of normal and reported serious bleeding of 1.8 per cent and minor hemorrhages of 3.4 per cent. Barker[3] started administering dicumarol on the second postoperative day and kept the prothrombin time between 10 and 30 per cent of normal. He reported major bleeding of 2.7 per cent in abdominal hysterectomies and 4.9 per cent in other operations.

In 1949, Wise and Loker[13] advised conservative dicumarolization, keeping the prothrombin time at 40 per cent of normal. They had no major hemorrhages and only 2 per cent of bleeding tendencies. It was found that fresh whole blood reduced the prothrombin time rapidly, but only for 10 to 12 hours, and vitamin K usually returned the prothrombin time to a safe level in 10 to 18 hours in patients with adequate liver function. Many excellent reports on dicumarol therapy appeared. Felder[5] stated that anticoagulants reduced the incidence of secondary embolism from an expected 30 per cent to 2.17 per cent and reduced the rate of secondary fatal pulmonary embolism from an expected 25 per cent to zero.

Therapy with the coumarin compounds is still undergoing the test of time and numbers. Newer methods of combating the problem of postoperative thromboembolism are constantly being introduced. To date we do not have the final answer, and the status of our ideas of the blood clotting mechanism is ever changing. Therefore, we must approach the problem of postoperative thromboembolism with an open mind.

Our regime for the prevention of postoperative thromboembolism complications consists of 13 salient points:

1. The proper preoperative preparation of the patient for surgery, including reduction of weight in the obese and treatment of any co-existent medical or mental condition.
2. Sensible preoperative and postoperative sedation to avoid prolonged narcosis and depressed cardio-respiratory functions.
3. Proper hydration of the patient to prevent hemoconcentration and dependent edema.
4. Gentle handling of the tissues during surgery and the insistence of pin-point stasis.
5. Thorough hemostasis to prevent hemorrhage, bloody drainage, and hematomas.
6. Simplification of the surgical procedure to avoid unwarranted prolongation of anesthesia and surgical exposure of the patient.
7. Application of elastic bandages to the extremities when indicated and the avoidance of constricting dressings to the abdomen.
8. Prevention and energetic treatment of postoperative abdominal distension.
9. Proper and frequent changing of the patient's position to avoid prolonged dependency of a part and the prevention of kinking of the popliteal and iliac veins.
10. Systematic postoperative exercises, including deep breathing and movements of the extremities.
11. Early postoperative ambulation.
12. Venous ligation when indicated.

13. The use of prophylactic anticoagulant therapy in selected patients.

The present studies were made between 1948 and 1954 on 4,383 surgical patients, including 432 herniorrhaphies and 387 hysterectomies. From the total number of patients, 331, or 7.55 per cent, received dicumarol. Dicumarol prophylaxis was administered to 326 patients postoperatively, and five patients were given dicumarol as part of their therapy for postoperative thrombophlebitis.

It is neither rational nor practical to give all patients dicumarol postoperatively. The indications and contraindications for dicumarol prophylaxis have been crystallized by experience. There are 12 types of patients who are prone to develop postoperative thromboembolic complications. They are: (1) cancer patients, (2) obese patients, (3) patients who have had extensive pelvic or vascular surgery, (4) debilitated patients requiring prolonged bedfastness, (5) patients who have had major gastrointestinal surgery, (6) patients with fractured hips, (7) those patients having surgery for large recurrent inguinal, femoral or ventral herniae, (8) patients suffering from vascular trauma or disease, (9) patients with certain blood dyscrasias, (10) patients who, through necessity, are receiving prolonged sedation and narcotics, (11) patients having partial vascular occlusion due to abdominal pressure, edema, large tumors, or obstruction, and (12) patients with a previous history of thromboembolic disease.

Any patient who fell into two or more of the above categories was routinely put on dicumarol prophylactically after surgery. Other patients received dicumarol at the discretion of the surgeon.

The 331 patients who received dicumarol postoperatively were put on the schedule outlined below, which we believe to be both safe and effective in preventing postoperative thromboembolic complications.

A prothrombin time is determined as a per cent of normal on the third postoperative day, using the method devised by Quick. It must be emphasized that the prothrombin time determination must be standardized, accurate, and reliable. Fortunately, most hospital laboratories meet these requirements by having well-trained technologists who are frequently and consistently running prothrombin time determinations. If the prothrombin time is between 90 and 100 per cent of normal on the third postoperative day, the patient is given 200 mg. of dicumarol. If the prothrombin time is between 60 and 90 per cent of normal, the patient is given 50 to 100 mg. of dicumarol. If it is below 60 per cent of normal, no dicumarol is given. After the initial dose of dicumarol is given, additional dicumarol administration is individualized for each patient. Daily prothrombin time determinations are performed to determine the patient's response. A prothrombin time of 40 per cent of normal is maintained through the 18th postoperative day in the average case. Selected patients are continued somewhat longer due to debility, prolonged bedfastness, or vascular disease.

Patients having purpuric diseases, bleeding lesions, advanced renal or hepatic disease, subacute bacterial endocarditis, or recent surgery on the central nervous system generally are not placed on a dicumarol routine for well-known reasons. If the prothrombin time falls between 20 and 40 per cent of normal, watchful waiting is recommended unless bleeding tendencies are present. If the prothrombin time is below 20 per cent of normal, or if there is some evidence of bleeding, large doses of synthetic vitamin K are given. If bleeding is severe, immediate blood transfusions and vitamin K are both administered.

RESULTS

The type and number of patients who received

TABLE I

Type of Surgery	*Total Cases*	*Number of Cases of Dicumarol*	*Per Cent of Cases of Dicumarol*	*Per Cent of Postoperative Thromboemboli*	*Per Cent of Postoperative Bleeding*	*Deaths*
Inguinal Hernioplasty	352	71	23.0	0.286 (a)	0	0
Ventral Hernioplasty	64	24	37.5	0	0	0
Femoral Hernioplasty	16	6	37.5	0	0	0
Varicose Vein Surgery	143	15	10.5	0	0	0
Hysterectomy	387	111	28.9	0.256 (b)	0.256 (c)	0
Cholecystectomy	257	37	14.4	0.778 (d)	0.389 (e)	0
Bowel resection with anterior anastomosis	49	11	22.4	0	0	0
Gastrectomy	91	15	16.5	0	0	0
Abdomino-perineal resection	59	26	44.1	0	1.69 (f)	0
Bowel Obstruction	10	2	20.0	0	0	0
Total	1428	326		3	3	0

a. One patient not on dicumarol developed thrombophlebitis in left femoral vein. No further complications after dicumarolization.
b. One patient not on dicumarol developed phlebothrombosis in left popliteal vein. No further complications after dicumarolization.
c. One patient developed moderate vaginal bleeding with a prothrombin time of 27 per cent of normal. Responded well to vitamin K.
d. Two patients not on dicumarol developed thrombophlebitis in lower extremity. One patient developed a pulmonary embolus.
e. One patient developed ecchymosis and transient hematuria which responded to vitamin K.
f. One patient developed moderate bleeding from the abdominal wound with a prothrombin time of 22 per cent of normal. Patient responded to vitamin K therapy.

dicumarol prophylactically and our results are given in Table I. The 71 patients who had inguinal hernioplasties and the 37 who had cholecystectomies were given dicumarol after exhibiting at least two of the forenamed indications for dicumarol prophylaxis, usually obesity or a previous history of thromboembolic disease.

In all, 4,383 surgical operations have been performed in the last six years with only one incident of a clinical postoperative pulmonary embolus and without any major postoperative hemorrhage. There have been four instances of clinical postoperative thromboembolic complications, and these patients were not on a dicumarol prophylaxis regime. One patient developed a left popliteal thrombophlebitis on the 4th postoperative day following a cholecystectomy. One patient developed thrombophlebitis in the right superficial femoral vein, followed by a right basilar pulmonary embolus five weeks after a cholecystectomy. One patient who had had a total hysterectomy developed a left popliteal thrombophlebitis on the 11th postoperative day. The other instance of postoperative thrombophlebitis occurred on the 5th postoperative day, following a right inguinal hernioplasty. Three patients recovered on heparin and dicumarol therapy without developing a clinical pulmonary embolus. The other patient who developed a pulmonary embolus made a complete recovery on the same therapy.

One patient showed moderate vaginal bleeding on the 5th postoperative day, following a total hysterectomy, and had a prothrombin time of 27 per cent of normal. Another developed moderate bleeding from the abdominal wound on the 7th postoperative day with a prothrombin time of 22 per cent of normal. Both promptly responded well to large doses of synthetic vitamin K.

Two patients developed prothrombin times of less than 5 per cent of normal without evidence of bleeding. One patient had a total hysterectomy for an adenocarcinoma of the uterus with a co-existing active bilateral thrombophlebitis from recent vein injections. This patient was operated upon with a prothrombin time of 70 per cent of normal, and postoperatively she was maintained with a prothrombin time of 40 per cent of normal without incident.

DISCUSSION

Modern improved anesthesia, preoperative preparation, surgical technique, and postoperative care have reduced the surgical and postsurgical mortality to a point where the surgeon now focuses a great deal of his attention on those problems that formerly received little consideration. One of these problems is that of postoperative thromboembolism, which fortunately is responsible for only a fraction of a per cent of postoperative mortality and morbidity. However, it is distressing to see a patient free of shock and infection, following surgery, suddenly develop a postoperative thrombus or die without warning from a sudden pulmonary embolus. Such instances remain vivid in the minds of those who practice surgery, and therefore a continuous energetic effort is being made to prevent such tragic complications.

Dicumarol has a definite place in the prophylaxis of postoperative thromboembolic complications. It has the advantages of oral administration, it is moderate in expense, and it can be given to the ambulatory patient with reasonable safety. However, anticoagulant therapy is but a part of the necessary regime to lower the morbidity and mortality from postoperative thromboemboli. The factors of gentle handling of the tissues, early ambulation, and the simplification of surgical procedures are vitally important in the prevention of thromboemboli.

The future may hold the solution to the problem of postoperative pathological blood clotting through the use of new techniques and new drugs. However, until such techniques and drugs prove their worth by extensive research, the surgeon cannot afford to apply them in private practice. In the light of our present knowledge we have shown that dicumarol, if used judiciously, is a valuable drug in the prophylaxis of postoperative thromboembolic complications.

SUMMARY

1. Six years of experience in private surgical practice using dicumarol prophylactically against postoperative thromboembolic complications has been presented.

2. A regime for the prevention of postoperative thromboemboli has been outlined, and the results have been tabulated, including the maintenance of a prothrombin time of 40 per cent of normal which has proved to be both effective and safe.

3. A total of 4,383 surgical procedures have been performed with only one instance of clinical postoperative pulmonary embolus and a 0.069 per cent incidence of postoperative thrombophlebitis.

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Salicylate Poisoning

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Ever since the first therapeutic use of the naturally occurring salicylates by Hippocrates some 2,400 years ago, they have probably been one of the most widely used therapeutic aids to mankind. Although the salicylates are widely and for the most part beneficially employed, they are not so completely innocuous as most would believe. While it is true that salicylates do not cause a great percentage of the death rate due to poisons, it is more important that most of the victims are in the pediatric age group. It is therefore the purpose of this paper not so much to reveal new data in the study of salicylate poisoning but merely to bring to the fore the fact that these relatively benign drugs can also be quite dangerous. The following is a brief résumé of a case seen at Children's Hospital, Washington, D.C.

A nine-months-old white male was relatively well until three days prior to admission, at which time the child began to show signs of an upper respiratory infection with associated nausea and vomiting. The baby was seen by a local physician, and the parents were told the patient had an inflamed throat for which penicillin was given. Aspirin was prescribed for the fever. As the baby would vomit shortly after the aspirin was given, the mother gave the child more aspirin, so that during a 36-hour period a total of about 37 grains was administered. The vomiting persisted and the child became progressively more listless and flaccid. On the day of admission, the respirations became rapid and labored, and the infant was brought to the hospital in a semicomatose state.

Physical examination revealed a well developed, well nourished, white male infant with apparent tachypnea and respiratory distress with marked sternal retraction. Temperature was 105, pulse 140, respirations 60. The head was asymmetrical with flattening of the left parieto-temporal region. The mucous membranes of the mouth and pharynx were considerably dehydrated. Examination of the heart and lungs was essentially negative. The only other abnormal physical finding was a hypospadias.

The initial laboratory work showed the following significant results:

Blood CO_2	21 volumes per cent
Blood Chlorides	113 mEq/l
Blood pH	7.47

The patient was placed on intravenous fluids immediately and subsequently received 75 cc. of Ringer's lactate, 280 cc. of 1/6 M. lactate, and 80 cc. of plasma with 8 cc. of concentrated sodium succinate. From the afternoon of admission to 3:15 A.M. the following day, the infant received a total of 500 cc. and from 3:15 to 5:15 A.M. another 450 cc.

The final CO_2 combining power was 20 volumes per cent and the blood pH was 7.1. The child expired at 5:15 A.M., some 14 hours after admission.

Since the child was hospitalized for only 12 hours, the case was turned over to the coroner for autopsy. A report was not available at the hospital, and the only information the author could obtain at this time was that the primary cause of death was acute cardio-respiratory failure secondary to salicylate poisoning.

In discussing the mode of action of the salicylates in causing the effects seen with toxic doses, it is deemed advisable to consider the pharmacology of the salicylates first, and thus a true understanding of their wide range of action on many organ systems may be appreciated.

Although the salicylates have long been used for empiric relief of such afflictions as headaches, myalgia, and arthralgia, their exact mechanism of action is not

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entirely known. It is suggested by Goodman and Gilman[1] that it is probably due to a central depressant action on the optic thalami. That this is the site of action rather than a cortical one seems to be substantiated by the fact that analgesic doses of salicylates cause no mental disturbances, anesthesia, or changes in sensation other than pain sense.

The mechanism of action in salicylate antipyresis seems to be a debatable issue with the two main theories being increased heat loss and decreased heat production. However, the preponderance of evidence seems to favor increased heat dissipation.[2]

The effects of therapeutic doses on the cardiovascular system are insignificant. However, with larger doses the peripheral vessels do show a tendency to relax, due to a direct effect upon the smooth muscle of the blood vessel. With toxic amounts there is a circulatory collapse caused both directly and by central vasomotor paralysis. In experimental work using perfused hearts, the concentrations necessary to injure that organ were much higher than are obtainable in man even with toxic doses. There has been no evidence as yet, either experimentally or clinically, that the salicylates, especially aspirin, are injurious to the heart.

Epigastric distress, nausea, and vomiting are the most common manifestations of the effect of salicylates on the gastrointestinal tract. Large doses have been reported to cause stomach ulcerations experimentally, and melena has been produced in the human being. Vomiting after toxic doses results from a combination of a local irritative effect and a central emetic action.

The salicylates also cause an increased flow of bile, probably by direct action on the liver cells. The experimental evidence concerning this, however, is under considerable dispute, and its use as a choleretic is not indicated.

In considering the metabolic actions of the salicylates, a prominent effect of therapeutic doses is the increase of urinary excretion of uric acid. The increase varies from 30 to as much as 100 per cent. The blood uric acid level may be reduced to one-third its original value. This is not always the case as the blood level may occasionally be elevated. The mechanism by which this increase in excretion is accomplished is not fully known. One of the more feasible theories is based on the inhibition by the drug of uric acid reabsorption by the renal tubules.

The number of red and white blood cells does not seem to be significantly altered by salicylate medication. The mode of action in depressing the leukocytosis in rheumatic fever is not well understood. Methemoglobin is not produced by the salicylates, and no reports of agranulocytosis from their use have been seen.

Moderate doses of salicylates seem to cause diuresis. This is probably best explained on the basis of a salt effect. Larger doses, however, decrease urine output, probably by actual renal impairment as attested to by decreased P.S.P. excretions and albuminuria. The term "salicyl" edema has been applied to the increase in weight seen in people on large doses of salicylate; however, no actual pitting edema is evident.

The anti-rheumatic actions are well known but just as poorly understood. Reduction of pain, immobility, and inflammation in the rheumatic fever joint is so dramatic that this point has been used by many as a diagnostic measure. However, the effect of the disease on the heart and other viscera is little altered, nor is the course of the disease.

The salicylates are readily absorbed from most body surfaces, including the skin, as well as the mucous membranes of the gastrointestinal tract. Considerable absorption takes place in the stomach. This probably accounts for toxic doses being administered unwittingly to vomiting patients. They are distributed to every tissue of the body without concentration in any particular tissue.

The salicylates are eliminated from the body by all channels, especially the urine. They can be demonstrated there from 10 to 15 minutes after administration of a therapeutic dose. About 20 per cent of the salicylate is destroyed by the tissues. Administration of sodium bicarbonate simultaneously does not hasten excretion of salicylate.

The dosage of the salicylate depends to a large extent on the condition being treated. The usual dosage for adults varies from 5 to 15 grains which may be repeated in 4 hours. For children a good rule of thumb is a grain for each year of age up to 5 years. In rheumatic fever, however, as much as 1.0 gm. should be given every hour until early toxic symptoms appear.

In discussing salicylate poisoning per se, some of the more salient features concerning incidence will be mentioned.

INCIDENCE

From the records of the U. S. Bureau of the Census,[2] it is apparent that there is no significant trend in the death rate from salicylate poisoning. The rate of salicylate poisoning is approximately 50 per cent higher in men than women. This is probably due to the ingestion of methyl salicylate as a substitute for alcohol in intoxicating beverages.

The average annual death rate is not high. During the period 1939 to 1943, with an annual mortality of 1,267 from accidental poisoning by all solids and liquids, salicylates contributed only four per cent of this total. This appears significant in light of the

fact that the drugs are in universal use and are easily procured by the general public.

Deaths due to acetylsalicylic acid represent approximately one-third of the mortality from all salicylates. Here again males predominate with a 20 per cent higher incidence than females.

There are significant differences among the various age groups. There is a relatively high mortality rate in children between the ages of 1 and 4 years. The mortality rate drops to a low level in the age class 5 to 14 years and then rises in the succeeding age groups. The high rate in the age class under 5 is probably due not so much to increased toxicity for children but rather to medication by ignorant parents and the propensity of children to swallow any readily available object.

From 1933 to 1943, the U. S. Bureau of the Census recorded 224 deaths due to methyl salicylate in children under 5 years of age and an average annual mortality rate 25 times higher than that of the remainder of the population. This is probably best explained in that this compound is tempting to children since it has the odor of wintergreen flavoring so familiar in candy and soft drinks.

SYMPTOMS

The time interval before onset of symptoms depends on the method of ingestion, that is, whether it is administered accidentally or in therapeutic doses. On the average, about 12 to 24 hours are required before symptoms appear when the drug is taken accidentally in toxic quantities. However, when salicylates are taken in divided doses, toxic symptoms do not appear for from 1 to 4 days.

The symptom most commonly seen is hyperpnea. At first it is mild, then it becomes more severe, and finally the stage characterized by Kussmaul breathing is reached. This type of respiration continues until either the acidosis is treated properly or the patient dies of respiratory failure. There are, in addition, numerous other signs and symptoms such as: vomiting, which may contain coffee ground material; an acetone odor to the breath; irritability and restlessness; cyanosis or pallor; semiconsciousness or stupor; profuse sweating; dehydration; fever of high degree; convulsions, or abdominal pain. Occasionally there may be actual bleeding, or a purpuric rash may be present.[8] Sometimes renal involvement is evidenced by scanty urine. This may progress to anuria as intoxication increases. It is fairly common for adults to complain of tinnitus and dizziness. It is also not uncommon to see mental confusion and delirium.

LABORATORY FINDINGS

The significant laboratory findings vary, depending on the degree of toxicity and the time or stage at which the studies are made. Probably the most common finding is the presence of salicylates in the blood and urine. The amount of salicylates required in the blood to produce toxic manifestations varies with the individual. A blood level as low as 29 milligrams per cent was found in one patient with symptoms, while another with a level of 68.4 mg. per cent recovered after proper therapy. The blood sugar may be reduced to as low as 44.8 milligrams per cent in patients with toxic complaints. In the uncompensated acidotic stage, the carbon dioxide combining power is almost always decreased substantially. Some reports have shown a CO_2 as low as 14 volumes per cent. A blood pH of 7.1 has also been reported.

The prothrombin time is often increased. The highest reported is two minutes and forty-five seconds. The longest bleeding time recorded to date is 10 minutes, with a clotting time of 7½ minutes.

The reaction of the urine usually depends on the stage of intoxication. At first there is usually an alkaline reaction, but later, when acetone and diacetic acid are formed, the urine becomes acid.

PATHOLOGIC PHYSIOLOGY

In 1937, Dodd et al.[4] reported the effects of salicylates on experimental animals. The direct effects of the drugs are an increase in the metabolic rate with an increase in heat dissipation, nausea, vomiting, irritation of the kidney, and more or less mental disturbance. The vomiting, diuresis, and sweating all act to produce dehydration with its subsequent rise in temperature and decrease in urinary output.

Acidosis results mainly from production and accumulation of ketone bodies and inadequacy of circulation. Ketosis is caused by depletion of food reserves of the body incident to the rapid metabolic rate.

Dodd[4] has stated, "No serious symptoms are produced by large amounts of salicylates experimentally, so long as fluid intake is adequate and there is no interference with heat loss." However, it was Hartmann et al.[5] who clearly outlined the process of excess salicylates in the human.

1. *Early effects:* There is hyperpnea due to stimulation of the respiratory center directly by the salicyl radical. This produces a respiratory loss of carbon dioxide, altering the sodium bicarbonate-carbonic acid ratio in the direction of alkalinity. This is the stage of respiratory alkalosis.

2. *Later effects:* The next stage is one of compensated acidosis caused by ketosis resulting from altered carbohydrate metabolism and the presence of retained acid anions in the blood and tissue fluids. The alkali reserve has been lowered previously to

compensate for respiratory alkalosis. Compensation is now maintained by renal and blood buffer systems and by the respiratory mechanism. The hyperpnea, which is now present, is due to the central effect of increased acidity of blood and tissue fluids.

3. *Late effects:* The final stage is that of decompensated acidosis with depletion of the alkali reserve, decrease in blood pH, and failure of the respiratory center in its attempt to achieve compensation.

More evidence of disturbed physiology is the increase in the prothrombin time by large doses of salicylates. Stevenson,[10] in reviewing 13 autopsies in cases of salicylate poisoning, found that one-half of the cases showed petechiae of the epicardium and one-third showed hemorrhages in the pleurae and lungs.

In experimental work with convalescents, Fashena and Walker[9] found a marked prolongation of the prothrombin time with doses of sodium salicylate ranging from 0.20 to 0.22 grams/kilo. This was noted to occur within the first 24 hours and reach a peak by the second day. Then followed a drop in the prothrombin time which, however, never did reach the level present before treatment. They consider the hemorrhagic complications to be due to a hypoprothrombinemia and other factors such as vascular dilation or vascular injury. Dietrick et al.[7] reported one case with a prothrombin time of 55 seconds and another of 25 seconds. Ashworth and McKenie[8] are of the opinion that these hemorrhagic manifestations are not based on liver damage because they can be altered by vitamin K.

PATHOLOGY

A common post-mortem finding is petechial hemorrhages. They are widespread involving brain, lungs, pleura, heart, and gastrointestinal tract. Occasionally they are seen in the adrenals, thyroid, spleen, and lymph nodes. The liver often shows parenchymatous degeneration, fatty infiltration, acute inflammatory changes, or hydropic degeneration with beginning necrosis. In some autopsies the kidneys were described as showing hyperemic changes and fatty degeneration.

TREATMENT

The mode of treatment depends for the most part on the stage the patient is in when seen. In accidental ingestion the first treatment is gastric lavage and hydration. Salicylates are usually found in the blood 10 minutes after ingestion and in the urine in 10 to 20 minutes. On the average, maximum blood levels are reached in an hour, with variations up to 12 hours. After hyperpnea is evident, gastric lavage is of no use. During the first stage, that of alkalosis, glucose in saline should be administered, preferably intravenously, but the subcutaneous route may also be used. The purpose of this is to spare the body reserves of glycogen and combat alkalosis by providing chlorides to combine with base. The intravenous fluids should be continued as long as the patient cannot take fluids by mouth. This provides sufficient fluids to maintain renal excretion. Another important measure at this time is to give vitamin K, 10 to 25 milligrams, and vitamin C, 100 mg., in order to combat the hypoprothrombinemia and bleeding which will otherwise ensue. This first stage is usually determined by observing the carbon dioxide combining power and the presence of an alkaline urine.

The second stage, that of compensated acidosis, is best treated with glucose solution and alkalizing agents. One of the most widely used alkaline solutions is the 1/6 molar lactate. Ringer's lactate may also be used. The amount given depends mostly on the value of the carbon dioxide combining power. Even so, if this cannot be determined, the degree of acidosis should be estimated and the 1/6 molar lactate given. The solution of 5 per cent glucose supplies necessary carbohydrate, aids diuresis and hydration. If the response to the lactate solutions does not appear adequate, a 1.5 per cent solution of sodium bicarbonate may be given intravenously. Some reports have shown excellent results with this agent. If the patient can take fluids by mouth, orange juice and sodium bicarbonate may be given. Vitamins K and C should still be given subcutaneously, however. An oxygen tent will usually prove to be necessary and transfusions of whole blood may be given, especially in the presence of widespread hemorrhage. Adequate fluids to help in eliminating the salicylates are very important.

Whether the third stage is present is difficult to determine; here a blood pH often helps. The treatment is the same as for the second stage but should be intensified. It is important to keep in mind that therapy should never be relaxed until the patient is conscious, well hydrated, and has adequate renal function.

If convulsions ensue, sodium phenobarbital should be used and a lumbar puncture may be carried out.

In conclusion it might be said that it is well to suspect salicylate poisoning in any hyperpnea of unexplained etiology. As a practical diagnostic measure, Gerhart's test may be used. It simply entails adding ferric chloride to the urine. A purple interfaceal ring develops which persists on boiling.

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Laparotomy Incisions

Barrett A. Nelson, M.D.

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Selection of a particular incision for a given laparotomy is probably very largely a matter of habit. Most older surgeons are still using the same incisions with which they started. Younger surgeons usually follow the techniques of those under whom they have trained. This is interestingly noted in such an outstanding group as the Lahey Clinic where Lahey used rectus muscle splitting incisions. Cattell still defends that procedure, though he admits the incision must not be too long or extend too low in the abdomen.

In careful studies comparing types of incisions, almost invariably data are presented supporting use of transverse incisions. On canvassing my own surgeon friends, I find that a large proportion give the matter little or no thought. They consider it of small importance and blithely continue their time-tried incisions that seem to serve their purposes. Those who have seriously considered the matter to the point of comparing various techniques almost invariably trend toward adoption of the transverse incision. In spite of this, I believe there are still many more vertical than transverse incisions.

The transverse incision is not at all new. It was 131 years ago, in 1823, that Baudelocque described a low transverse incision for cesarean section. Pfannensteil presented his well known incision in 1900. Frederick Coller of Michigan is one of the most active of contemporary exponents of the transverse incision and presents his case most ably and convincingly.

It is difficult to build a case against the transverse incision. Proponents set forth the transverse direction of the aponeurotic and fascial fibers; the transverse course of the nerves that traverse the abdominal wall; the increased ease with which a transverse incision may be closed; the greatly decreased disrupting forces of coughing, sneezing, retching, and ordinary muscle activity as applied against the sutured transverse wound; finally, the tremendously greater and more satisfactory exposure obtained through the transverse incision greatly facilitating the operative procedure.

It is quite obvious that bites of suture across the horizontally directed fascial fibers must give immeasurably more strength to a wound than those placed in the edges of a wound which has cut across those fibers, as is true in vertical incisions. It is like comparing the holding power of a nail driven into the side of a 2 x 4 with that of one driven into the end grain.

How many times has each of you sweated it out trying to pull together the edges of a vertical wound in an obese patient with distention, while the anesthetist has permitted her to waken to a straining stage. Think of the many times your suture has pulled out of the fascia leaving a frayed, shredded margin that you finally got together, reinforced with large retention sutures, possibly over buttons or short pieces of rubber tubing; you applied 3 to 5 yards of wide adhesive tape, finally a tight muslin binder, and cautioned the nurse not to let her strain or sit up for at least a week. And then if it did not disrupt, you shook your head and wondered why.

It is many times easier to close the transverse wound. You get a firm bite across the fascial fibers. It is almost impossible to pull it out; the margins simply can't get away. They come together easily and are firmly held there. Furthermore, any straining movements of the patient have comparatively little effect as interference to your progress. In 1927, G. A. Sloan did 20 laparotomies using L-shaped incisions. With the patient under light anesthesia he applied spring balances to the wound margins and measured the forces required to hold the margins together

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From the Nelson Clinic, Manhattan, Kansas.

against the patient's active respiratory movements. To hold vertical incisions together took a force 30 times greater than for transverse incisions. Furthermore, he found the required force increased in proportion to the square of the length of the wound. In one of the longer vertical wounds, the required force was 45 times that for an equivalent transverse wound.

It is well known that if there is division of more than one of the horizontally directed nerves segmentally supplying the rectus muscle, the fibers of the muscle medial to the incision atrophy and are thereafter largely useless, furnishing neither support nor muscular action. The same is true of the flat muscles lateral to the recti. There seems to be little argument against the advisability, from this standpoint, of placing an incision between nerve trunks rather than cutting across them.

Finally, when one sees the gall bladder pop up through a transverse incision like a bass trying to jump into the boat, he wonders why he struggled and perspired while using a split rectus incision all those years. And gastric surgery is right on top with a wide curved transverse incision. It is true that occasionally there must be a short vertical limb added with a high, narrow, subcostal arch, but only rarely even then.

Possibly a common hesitancy toward transverse incisions is the natural feeling against cutting across the rectus muscle. There need be no fear on this score. While such an incision is catastrophic in the long muscles of the extremities, where the nerve supply is thus completely severed, the nerve supply to the rectus muscle is segmental and enters from its lateral border. Transection of the rectus severs no motor nerves. Muscle splitting does. And it is quite unnecessary to suture the ends of the severed recti. There is almost no retraction, especially in the upper abdomen, as the tendinous intersections firmly hold the muscle to the anterior sheath. On opening such a wound it is often almost impossible to demonstrate the point of muscle division. Even where the abdomen is opened soon after closure, muscle healing is quite amazing.

In the lower abdomen, Cherney transects the fibrous tendinous parts of the recti. These, of course, are readily re-united by firm suturing.

For cholecystectomy we use a transverse incision extending from the midline to a point anterior to the lower costal cartilages. The right rectus is transected. The lateral portion of the superficial wound is pulled downward slightly, and the muscles lateral to the rectus are split apart. In one-third to one-half of the cases the obliques and the margin of the latissimus dorsi are also transected for a short distance. No ligatures are used. All bleeding is quickly controlled by electrocoagulation. The posterior rectus sheath and peritoneum are opened transversely. Excellent exposure results, even in the obese patient. Closure of the fascial layers of the lateral muscles up to the margin of the rectus is done with running sutures of chromic 0. The rectus is wide at this level, so the posterior rectus sheath forms the main part of the closure. This is firmly accomplished with figure 8 sutures of 00 silk or heavy cotton, catching fascia and peritoneum. The same sutures are placed in the anterior sheath, where wire may be used if desired. No sutures are used in the muscle. The superficial fascia is closed with interrupted sutures of fine nylon. A subcuticular suture of 000 plain catgut is placed in the skin. No retention sutures are used, no large strips of adhesive; no binders are applied.

For gastric or any sub-diaphragmatic surgery, or any extensive surgery of the upper abdomen, we very much like a curved incision, convexity upward, starting from the costal margin laterally, curving across a short distance below the xyphoid process, and continuing clear across to a corresponding point on the other side. Both recti are transected. Lateral muscles are split apart, to some extent incised. Exposure is gratifying. Some of these incisions are 18 inches long, and the patient looks as though he had committed hari-kari. Infrequently a narrow, high arched patient gives a better view with a short midline extension upward. Closure is the same as in the cholecystectomy incision, starting with two firm figure 8 sutures in the midline. Again, no retention sutures are used.

For a number of years we have standardized our appendectomy approach, using a transverse incision starting in the midline approximately at the level of the anterior superior spines. It passes laterally, curving slightly upward following the lines of Langer. It is carried through skin, superficial fascia, and anterior rectus sheath. In this instance the rectus is not transected but is freed from the anterior sheath, retracted medially along with the inferior epigastric vessels, and held there by the assistant. The posterior sheath and peritoneum are incised transversely. Most appendices are easily reached. If one is found to be lateral or down behind the cecum, the incision is carried across the linea semilunaris, and the muscles are split laterally as far as necessary. Again, exposure is excellent. Closure is simple. A single strand of chromic 0 with a swedged-on needle is placed as a running suture in the posterior sheath, starting medially, continuing laterally to the muscle margin, back across the anterior sheath to the midline. Fine nylon is used in the superficial fascia, plain 000 subcuticularly in the skin.

Now I must make a confession. For pelvic surgery I still prefer the midline incision from the umbilicus to the pubic symphysis. And I mean clear

down between the recti to the pubic bone. If it goes clear down it is rarely necessary to go above or to the side of the umbilicus. It has seemed more satisfactory than either the Pfannensteil or the Cherney incision. Maybe it is just habit again, but I have switched back and forth a few times and always end up in the middle.

But the closure technique is extremely important. With proper care, this incision heals as strongly as any, but not unless certain details are scrupulously followed. First, the peritoneal margins must be everted to prevent adhesions to the underlying viscera. Waugh at Rochester stressed this years ago, and I bless him every time I re-open an abdomen and find a scar free from intra-peritoneal attachments. We use our swedged-on needle with chromic 0, placing a running, mattress, lockstitch. The rectus muscles are brought together in the midline with a loosely placed running suture of plain gut. This adds little, if any, strength, but it obliterates the dead space and keeps the anterior and posterior rectus sheaths apart. The anterior sheath is closed with mattress sutures of 00 silk, heavy cotton, or wire. This, we believe, is important. Catgut, running or interrupted, is inadequate. Simple sutures across the wound edges, or even figure 8's, invite shredding out and disruption. Only a mattress suture which ties around bundles of transverse fascia fibers can give a firm, safe closure. It is simply another case of avoiding a nail in the end grain. Once more the nylon is used, then the 000 plain subcuticularly.

That nylon and plain catgut subcuticularly require a little explanation. The former was introduced to me by Dr. Thomas P. Butcher of Emporia. It is nothing more nor less than Coates and Clark's black nylon thread, size A, purchased at the department store. Since I first tried it, I must admit that the price has advanced twice, 60 per cent, then another jump of 20 per cent. (By the way, there are 100 yards in each spool, so I suppose that even as much as 10 cents per spool is not exorbitantly expensive.) It is smooth; it is tough and hard to break; it is non-irritating to the tissues. I have never seen it spit out as cotton so frequently does. Being smooth, it requires a tight, firm, third throw on each knot. For tiny needles it requires a little more care in threading. The small French eye needles are convenient for this. But we like this nylon immensely for all sutures in the fat where catgut produces so much more reaction. We use it routinely for anastomoses of the alimentary tract, for hernia repairs, for any skin closure where catgut is inadvisable. We do firmly believe it makes for a much better healed wound if all dead space in the fat is entirely eliminated. This fine nylon is excellent for that purpose.

Practically all of our abdominal wounds have the skin closed with subcuticular 000 plain catgut. Several years ago I pondered on the routine skin closure of perineoplasties utilizing catgut. If so successful in that area, why not in a clean abdominal wound? Several sizes of chromic and plain catgut were tried. Chromic lasts too long. As long as 30 to 50 days later there is sometimes a marked reaction under the skin. The fine 000 plain is most satisfactory. A stitch is placed in the superficial fascia under the skin at one end of the wound, with the knot deep in the fat. This stitch is left long. A similar stitch is then started in the fat of the other end of the wound, the knot underneath, deep in the fat away from the skin. This suture is carried the length of the wound in the deepest layer of the skin. Each stitch is pulled tight as it is placed, and finally the last stitch is reversed, turned back one stitch, tied tightly to the short length previously placed there. The suture is pulled up so it may be cut close against the knot, which then snaps down under the skin and disappears. There is no penetration of the skin. Each stitch is carefully placed just under the deep edge of the skin. Bites must be short but fairly close together. If bites are too long or too close together, there may be necrosis of the skin margin. It is the deep portion of the skin only which heals strongly, so each stitch must be pulled up snugly. The superficial layers of the skin just fall together. In 5 to 10 per cent there will be a little serous ooze from between the skin edges at one end or the other (over the knots). This is of no consequence. Wipe it away with an alcohol sponge.

For postoperative dressing a narrow, light gauze strip is applied with ½-inch adhesive tapes three or four inches long. The gauze serves only to blot up the slight bloody ooze from the skin edges. It is removed on the first postoperative day; there are no dressings thereafter. Only the patient's shirttail is pulled down over the wound. The results? A wound which is unbelievably less painful to the patient than one where sutures penetrate through the skin; a wound that heals quickly, is dry and clean, and not sensitive to pressure, to clothing or to active motion; a wound that heals as a narrow line with no stitch marks; and—a joyful lighting up of the patient's face when you tell him, "There will be no stitches to take out."

Please note: retention sutures have been discarded entirely. No supporting large bands of adhesive tape or ABD pads are used (excepting drainage cases, of course). There are no abdominal binders. Our patient is ambulatory on his first postoperative day, on the operative day if he likes. And yet, with this technique as a routine in nearly 400 cases, we have had not one case of disruption of the wound, not one postoperative hernia, not one wound infection in any

clean case. Our appendectomy patients are usually discharged by the fourth day, most of those who have had cholecystectomies, hysterectomies and herniorrhaphies by the end of the week.

Most surgeons will think this too "puttery" a routine to bother with. Of those of you who do try it, many will be unwilling to spend the extra effort and time to continue it. Most of us are in a hurry. But if there be among you any of the finicky old spinsterish type who are willing to fuss a while over little details, I firmly believe you will eventually find it a gratifying closure routine.

Sunlight and Cancer

Herbert L. Ketterman

Kansas City, Kansas

In 1894 Unna[1] pointed out that prolonged exposure to sunlight might give rise to malignant tumors of the skin. Hyde,[2] writing in 1906, used statistics on the prevalence of cancer of the skin in various occupations and in various geographical locations to eliminate wind, heat, and frostbite, for example, as causes of skin cancer. He concluded: "It would seem that exposure to light and to light exclusively is the special stimulant provoking a certain proportion of the skin to the metamorphosis that means carcinoma."

In 1928 Findlay[3] reported that white mice manifested "papillomas and malignant epitheliomas" after eight months of daily three-minute exposures to radiation from a mercury vapor lamp. This work has been repeatedly confirmed with variations on mice and rats, using light from a mercury vapor lamp and sunlight with various lengths and intervals of exposure.

Blum[4] subjected albino mice to carefully measured doses of ultraviolet radiation which were given at regular intervals. After a considerable number of doses, tumors appeared on the ears of the mice, the time of appearance depending upon the size and frequency of the dose. Histopathological examination showed that the tumors were malignant and predominantly sarcomas. Grady, Blum and Kirby-Smith[5] as well as other investigators who have exposed mice to ultraviolet radiation have noted a much higher percentage of sarcomas than carcinomas.

Malignant tumors of human skin are nearly all epithelial, i.e., carcinomas in contrast to the ultraviolet radiation induced sarcomas of mice. The ultraviolet rays are virtually all absorbed in the epidermis of man, and it is here, where only epithelial cells are present, that human cutaneous cancer appears. The ultraviolet rays penetrate much deeper in the mouse to reach connective and other non-epithelial tissues, thus accounting for the high proportion of sarcomas.[6]

By use of appropriate filters it has been shown that the maximum carcinogenic rays are those of wavelengths between 2,600 and 3,000 angstrom units which lie in the same spectral region (ultraviolet rays) as those which produce erythema of "sunburn" and antirachitic effects of sunlight.[7] It is the short wavelengths (ultraviolet rays) of sunlight which penetrate tissue easily and produce their physiological effects slowly. The converse is true for wavelengths in the upper portion of the sunlight band (infrared radiation), whereas the central portion of the band has little known effect on the skin.[8]

The mode of action of ultraviolet light in the production of cancer is as yet not clear. However, it has been suggested that some disturbance in the proteins or nucleic acids might be responsible. This receives strong support from the fact that the absorption maximum of nucleic acids is about 2650 angstroms and that of proteins about 2800 angstrom units. This corresponds closely with the carcinogenic portion of the spectrum. It is to be expected, therefore, that various disturbances in these components might result from ultraviolet irradiation, and it seems not unlikely that such disturbances would have a profound effect upon cellular behavior.

Blum[9] has reviewed the pathology of acute solar erythema. Ultraviolet rays are chiefly absorbed by the cells of the horny and the malpighian layers; a small fraction may reach the papillary layer of the cutis. The molecular action of ultraviolet rays on the protein of the epidermal cells results in the liberation of vasodilator and leucotactic substances; these reach the cutis by diffusion after a significant time lag—the "incubation period" of solar erythema. Vasodilation, leukocytosis, and edema result in the corium, and

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cellular degeneration occurs in the epidermis. This stage is soon followed by a reparative reaction ending in restitution and hyperplasia of the epidermis, which is shared by all layers except the basal, and by absorption of the inflammatory exudate in the cutis. Melanization of the epidermis occurs and extends through all layers of the rete, often into the horny layer. With the exception of the pigmentary response, the process does not differ materially from any ordinary inflammatory reaction at any other site of the body.

Clinically, complete repair with pigmentation is usually attained; if, however, the epidermis has been completely destroyed, scarring ensues. The hyperplasia of the horny layer, which may persist up to two months, is chiefly responsible for the resistance against further irradiation shown by the recently sunburned. The cells of the epidermis are incapable of developing immunity against repeated solar trauma; after a variable interval, normal sensitivity returns even though pigmentation may still be marked.

The mechanism of chronic sunburn is less well understood. The frequent repetition of the stimulus of solar radiation apparently leads to an exhaustion of the reparative forces, and irreversible changes take place in the cutis. The changes affect at first the supporting and elastic fibers of the corium and the blood vessels; eventually epidermal changes supervene which result in atrophy, permanent freckling, areas of depigmentation, persistent desquamation, keratoses, and possibly carcinoma.

Many of these changes may appear in later life in the individual who has spent all or most of his life on farms, and it is the blue-eyed, blonde, and pink-skinned individual who is most susceptible. Early changes are a dryness and wrinkling of the exposed skin which become progressively more severe. Permanent freckling over the dorsal surfaces of the hands, cheeks, and ears is seen. Over a period of several months or years, small horny excrescences develop; these senile keratoses belong in the group of skin premalignancies. If not adequately treated, these senile keratoses continue to grow, and the underlying skin becomes infiltrated with a hard, nodular, pearly infiltrate which is already a squamous cell carcinoma.[10]

Carcinomas of the skin are divided into two main types, the basal cell carcinoma and the epidermoid carcinoma. The early basal cell carcinoma usually has a gray somewhat translucent appearance and may be present as a small nodule beneath the thinned out overlying epithelium. In large basal cell carcinomas, areas of yellowish necrosis are frequent. The epidermoid carcinoma, often keratinizing, may show yellowish-gray areas on cross section. The large epidermoid carcinoma with an ulcerated surface is heavily infected.

The evolution of the process of development of skin carcinoma varies. Fifteen or 20 years may be necessary to produce the carcinoma, but malignant lesions have occurred in a matter of a few months.

It has long been known that there are certain individuals who are highly susceptible to carcinoma of the skin. As has been mentioned previously, there is a much higher incidence in patients who work under strong exposure to the sun. In 200 cases studied by Roffo,[11] 97.8 per cent of the patients were farmers, field hands, bricklayers, seamen, etc., whose occupation necessitated exposure to sunlight over the years. The most frequent ages involved in this series were between 61 and 70 (29.7 per cent), whereas octogenarians accounted for only 8.7 per cent.

Another factor to take into account in the production of this process is the biology of the skin. It is indubitable that there exist skins with greater or lesser predisposition toward solar erythema, a predisposition connected with the absence of pigment. It is the white skin, especially of blondes, which seems to be most sensitive. The white person's skin is 10 times more sensitive to ultraviolet rays than the Negro's skin.[12]

Piers[13] reported on a series of 51 cases of cutaneous cancer he had seen between 1941 and 1947 in Kenya. Forty-five of these were basal cell carcinomas, the cheeks and nose being the most common sites. The ages of the patients ranged from 29 to 89 with one-third of the cases in patients between 60 and 69. Thirty-seven of the 51 patients gave a clear history of long occupational exposure, and in 34 patients chronic sunburn was present. He concludes that sunlight is the paramount factor in the causation of cutaneous carcinoma in Kenya.

Molesworth (cited by Piers[13]) stated in 1937 that cancers of the skin in Australia exceed in number those of all other organs, and that 99 per cent of these are due to chronic sunburn.

In a series of 5,000 cancer cases observed before 1928 at the Institute of Experimental Medicine for the Study and Treatment of Cancer in Buenos Aires,[11] the great frequency of cancer of the skin was notable. The regions most often attacked were as follows:

1. Carcinomas of the skin of the face	95.51%
2. Carcinomas of the dorsum of the hands	3.07%
3. Carcinomas of the scalp	1.02%
4. Carcinomas of the foot	0.40%

Furthermore, this relationship with intensity of solarization of the skin is attested even more if consideration is given to the frequency of carcinoma of the face, the nose showing the highest percentage as it is the most exposed part. The following summary gives percentages affecting the different parts of the face:

Nose	61.4%

Maxilla	18.0%
Forehead	7.7%
Ear	7.9%
Temple	4.0%

SUMMARY

Five major lines of evidence supporting the concept that sunlight is a causative factor in the production of skin cancer have been discussed. These lines of evidence are: (1) Cancer of the skin of laboratory animals (mice and rats) can be induced by exposure to ultraviolet radiation; (2) Cancer of the skin is more common among outdoor than indoor workers; (3) Cutaneous cancer is less prevalent in Negroes than in the white races, presumably because the former are less susceptible to sunlight; (4) The incidence of cancer of the skin is greater in regions of the earth that receive high insolation; (5) Cancer of the skin occurs principally on parts of the body most exposed to sunlight.

None of these lines of evidence is completely convincing in itself, but, as evidence accumulates, it converges in a striking manner to support the conclusion that sunlight is a major cause of cancer of the skin.

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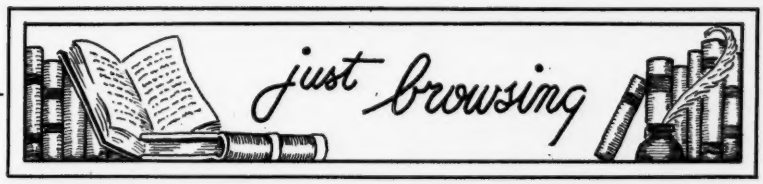
Although it is not the privilege of every physician to serve on the firing line through active participation in medical organization work, nearly everybody can serve in the splendid work being done by the American Medical Education Foundation and the World Medical Association. How can alumni of medical schools fail to recognize that it is their obligation to contribute according to their means to the American Medical Education Foundation and thus forestall government subsidization of medical schools and the ultimate socialization of the practice of medicine? What valid excuse can physicians have for not supporting the World Medical Association, which has been so helpful in cementing international physicians' relations and improving medical care throughout the world?

J. D. McCarthy, M.D., Chairman
A.M.A. Council on Medical Service



"Every operation in surgery is an experiment in bacteriology. The success of the experiment depends not only on the skill but also upon the care exercised by the surgeon in the ritual of the operation."

"In the transition of a patient from ill health to sound health the operation itself is only one —though it may be the most important—of all the factors concerned in this fortunate event."

"After this point [the incision of the skin] the use of antiseptics in a 'clean' case is rarely necessary, is often undesirable, and is almost always of greater harm than benefit. It is to insult tissues and to doubt them, when it would be better to trust their very considerable powers of self-defence."

"Surgery should be a merciful art. The cleaner and the gentler the act of operation, the less the patient suffers, the smoother and quicker his convalescence, the more exquisite his healed wound, and the happier his memory of the whole incident, to him probably one of the most important in his life."

"It would be well if surgeons the world over took a little more pride in the wounds they inflict. The appearance of a wound is often the best index to the quality of the work that has been done throughout the operation."

"It should be our ideal to complete such an operation [thyroidectomy], which in my student days was often one involving much loss of blood, without staining the towels round the wound."

"Throughout the operation there must be no undue exposure of parts. . . ."

"The most important person present at an operation is the patient. . . . It is our duty to make the operation as little disagreeable as possible for him."

"Every detail in every operation is of importance, and should be conceived, practised, and tested with unwearying patience by the operator himself, and by him in conjunction with all his assistants. In surgery, at least, success may well depend upon the scrupulous, exacting, and unceasing supervision and close scrutiny of every smallest incident of procedure."

"In all the movements of the surgeon there should be neither haste nor waste. It matters less how quickly an operation is done than how accurately it is done. Speed should result from the method and the practised facility of the operator, and should not be his first and formal intention. It should be an accomplishment, not an aim. And every movement should tell, every action should achieve something. A manipulation, if it requires to be carried out, should not be half done and hesitatingly done. It should be deliberate, firm, intentional, and final. Infinite gentleness, scrupulous care, light handling, and purposeful, effective, quiet movements which are no more than a caress, are all necessary if an operation is to be the work of an artist, and not merely of a hewer of flesh."

Do you know or can you guess the source from which these quotations were taken? Do they sound up-to-date? Are they not all pertinent to today's surgery? Could we not improve our own surgery if we could remember and achieve each of these principles?

For some it may be a surprise, but to others it will not, that all these quotations are from an address entitled "The Ritual of a Surgical Operation," given by Lord Moynihan* in 1920. Offered to the profession 34 years ago, they are still as appropriate as at that time and serve as an example of the vision, understanding, and art of a master surgeon. All that is old in medical literature is not obsolete; much is basic and is useful and apropos for an indefinite period of time. A perusal of the writings of the master physicians and surgeons of yesteryear will furnish us both information and inspiration.—*O.R.C.*

* First published in the *British Journal of Surgery*, 1920, and reprinted in a collection of "Essays on Surgical Subjects" by Berkeley Moynihan (W. B. Saunders Company, 1921).

PRESIDENT'S PAGE

DEAR DOCTOR:

As this is being written, the New Year appears to bring with it a few problems that I wish could be settled by making a few good resolutions. One of these merits special consideration.

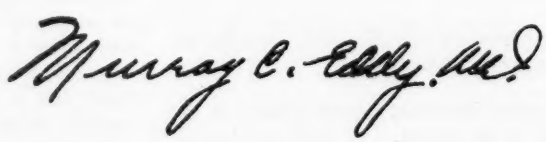
Your committee has had numerous meetings with a like committee representing the osteopathic profession in an effort to find a common foundation to support an adequate medical legislative structure. While these conferences have been candid in nature and at times quite heated in discussion, it cannot be said that there has not come from them an increased understanding of our common problems.

Throughout the entire negotiations the members of your committee have always maintained the same position, that we will actively oppose any legislation we feel is not in the best interests of the citizens of our state, and that we will propose, introduce, and actively support measures to improve medical care of our citizens.

Your Society will continue to press for increasing standards of medical training and medical care and will attempt as strongly as possible to so influence our legislators.

It is, therefore, up to you to give them your experience, your wisdom, and your most earnest advice in all matters pertaining to public health and medical care.

Yours very truly,



EDITORIAL COMMENT

Our New Cover Page

In January 1935, exactly 20 years ago, the JOURNAL presented its first distinctive cover page, without advertising, and including the now-familiar figure depicting a modern stethoscope and an original type stethoscope used by Laennec. This little figure was designed specifically for the JOURNAL as being symbolic of medical practice. Brad Thompson, the artist, was a personal friend of Dr. W. M. Mills, the editor at that time.

Twice since that date—in 1941 and again in 1942—the cover design was again changed, and each time this distinctive figure was incorporated in the new cover page, so that now it has become as much a part of the JOURNAL as any of the features on the inside and is something which is ours alone.

After 13 years without change, the little familiar figure has put on a new suit of clothes, and we are particularly pleased that again it was possible to have it designed by Mr. Thompson, now the art director of *Mademoiselle*. He was willing to do it once more, primarily because of his personal friendship for two former editors of the JOURNAL, Dr. Mills and Dr. Pyle. We are proud to present the JOURNAL to you in its new cover, first because we think it is attractive, then because it retains the now-traditional figure designed by Mr. Thompson, and finally because he has again helped to dress up our cover page. We of the Editorial Board are greatly indebted to "Brad" for his kindness in doing this work for his friends "back in the old home town," and want to take this opportunity to say "thank you" to him. We hope that all will like the new cover, and we believe that it is a step toward a better and more attractive JOURNAL.

Federal Aid in Kansas

PART IV

This portion of the report on federal grants to Kansas deals with the welfare program. As in previous sections, this is a factual review without editorial comment or opinion. The entire series, with material largely taken from research conducted by the Kansas State Chamber of Commerce, is for the purpose of informing the medical profession on some phases of governmental activity in Kansas. It should also be noted that the latest available figures are generally for the year 1953, so they do not necessarily reflect the present situation.

There are 11 federal grant-in-aid welfare programs in Kansas, all of which fall under one of the major categories listed below.

PUBLIC ASSISTANCE

The federal government provides funds for the operation of four public assistance programs. The Social Security Act of 1935 made three of them possible, and the fourth came into being following a 1950 amendment. Kansas participated in the three as of August or September 1937 and in the fourth as of October 1950. The money for all comes from the Department of Health, Education, and Welfare. These programs are administered in Kansas by the State Department of Social Welfare and at the county level by the 105 County Departments of Social Welfare.

The first of these is for Old-Age Assistance which offers financial aid to needy individuals over the age of 65 years who are not living in a public institution. Funds under this program are in addition to old-age and survivors insurance also paid by the Social Security Administration.

The payment formula has frequently been misunderstood. In each monthly grant the federal government pays four-fifths of the first $25 and one-half of the next $30, but nothing beyond those amounts. Also received from federal sources is half of county and state administrative costs.

There are 11 principal federal requirements which must be met before this program may be placed into effect. It must be in effect in all political subdivisions of the state, it must be administered by a single state agency, and the state must participate financially in the plan. Reports must be submitted, there must be a merit system in the state, clients must be given a right to appeal. Other income must be individually taken into account in determining need, there must be safeguards as to information, states may not make a higher age requirement than 65 years, no citizenship requirement may be made to exclude any citizen of the United States, and, finally, residency requirements may not exclude anyone who has lived in the state five of the last nine years and the last continuously.

Under Old-Age Assistance in 1953, Kansas received $14,298,939 from federal sources and spent $8,565,912 state funds and $5,200,000 county funds for a total of $28,064,851. This has increased every year in each category, now being almost double the 1947 figures.

The second public assistance category is Aid to Dependent Children, which provides financial assistance to needy children under 16 years of age, or 18 if they are in school, who are deprived of parental support by reason of death, absence, or incapacity of a parent.

Here the formula for federal aid goes as follows: Federal participation is four-fifths of the first $15

of each eligible child and of the relative with whom the child is living. Then one-half the remainder up to $30 is paid for the first child in the household and half up to $21 for each additional child in the same home. Again half of the state and county administrative costs are paid. In general, requirements are similar to those outlined above except for a change on age and a relaxation of residence regulations.

This program has been relatively constant in cost over the years, a little higher than in 1947, a little lower than in 1952. In 1953 the federal government gave $2,690,748, the state $1,387,078, counties $1,117,252 for a total of $5,195,078.

The third category is Aid to the Blind, and it gives aid to needy blind persons who are not living in an institution. The federal participation formula and the federal requirements are very similar to those applicable for Old-Age Assistance. The cost is far less and has remained nearly constant over the years. In 1953 the federal government contributed $240,004, the state $160,981, and the counties $99,896 for a total of $500,881.

The fourth category, Aid to Permanently and Totally Disabled, called Aid to Disabled in Kansas, was the most recent addition to the public assistance program, being started in 1950. It is available to needy persons 18 years of age or older, not living in an institution, who are disabled in mind or body to the extent that they are unable to follow any substantially gainful or useful occupation, trade, profession, or service. The formula and requirements governing federal participation are similar to Old-Age Assistance except that medical certification is necessary. In Kansas this program has grown since its beginning to 1953, when the federal government paid $1,130,245, the state $657,229 and local sources $554,021 for a total of $2,341,495.

CHILD WELFARE SERVICES

This program began in Kansas in 1936 and is federally administered through the Social Security Act of 1935. It is administered in this state by the Department of Social Welfare, and there is no local participation. Its purpose is principally for providing services rather than financial aid for dependent, neglected, or homeless children who are in danger of becoming delinquent.

The federal government annually appropriates up to $10,000,000 for this work, allots $40,000 to each state, and divides the remainder according to the relative rural population of children under 18 years. A further point is that this project is specifically designed for rural areas. There are no matching funds required, but the program is arranged on a contract basis.

In general, this program is expanding in Kansas from a total of $93,069 in 1947 to $287,241 in 1953. Last year the federal grant to this state was $121,257, and Kansas spent $165,984.

A second project under this heading is for crippled children's services, operated in Kansas since 1937. Federal participation is again through the Department of Health, Education, and Welfare. State administration is by the Crippled Children's Commission. There is no local agency involved.

The purpose is to locate crippled children and to provide corrective and other services for them. The program pays for medical care, hospitalization, and appliances. It conducts diagnostic clinics. The Kansas law makes children under the age of 21 years eligible if their parents are unable to pay either all or part of such necessary care. Harelip, cleft palate, congenital cataract, orthopedic deformities including results from poliomyelitis and from burns are representative conditions that may be cared for.

AID TO EDUCATION

There are numerous federal aid programs in the field of education, most of which were discussed last month. In addition to those are two welfare projects. The first is called Vocational Rehabilitation of the Blind and became operative in Kansas in 1943. At the federal level this is a part of the vocational education grant, but it is separated in Kansas where the Board of Vocational Education handles all but this portion of the work which is under the direction of the Board of Social Welfare.

In general, the federal government pays all the cost of administration, all the cost of vocational guidance and placement services, and half the cost of training, medical and hospital care, etc. The purpose is to prepare blind persons for economic independence. The program corrects all possible physical defects, trains the patient for employment, and purchases necessary equipment.

The cost has steadily increased since 1947 when the total for Kansas was $17,712 to 1953 when it was $65,552. For 1953 the federal government paid $46,200 and the state $19,352. There is no local participation.

The second program is a federal grant for Education of the Blind, operating in Kansas since 1879. The Office of Education administers this at the federal level and the Board of Regents locally. The service is to prepare and distribute books in Braille and other teaching materials for blind persons. Figures for 1953 are not available but in 1952, $1,282, all from federal funds, was spent in Kansas.

HOME FOR DISABLED VETERANS

Since 1895 federal funds for veterans' care in a specialized home have been available through the

Veterans Administration. The program is administered in Kansas by the Veterans Commission. The federal payment matches state funds at the rate of $500 a year per veteran cared for. In Kansas this is spent for the State Soldiers' home at Fort Dodge and, because of state legislative action in 1951, a considerable reduction has recently occurred. In 1953 the federal government paid Kansas $8,387, and state expenses are not known. In 1952 the federal grant was $12,569 and state support was $232,673, but some of this was spent for non-veteran care.

SUMMARY

The above then are welfare programs in which the federal government shares the cost of operation. Grouping all together as listed in this category, Kansas received $16,874,577 from federal sources and spent $17,808,546 in state and county money. The federal welfare appropriations to Kansas are 1.4 per cent of the total spent in this field among all states. The national federal cost is just under two billion dollars.

Help for the Afflicted

The progress of medicine during the past 20 years, star-studded with the miraculous discoveries of penicillin, radioactive substances and the like, not only leaves one a little breathless at times but inclines one to overlook an occasional therapeutic gem. Such a gem is the hand talking chart reprinted on this page, the result of the efforts of Dr. Hamilton Cameron to bring some small measure of comfort to the aphasic patient who needs desperately to communicate with his world before it spins away from him forever.

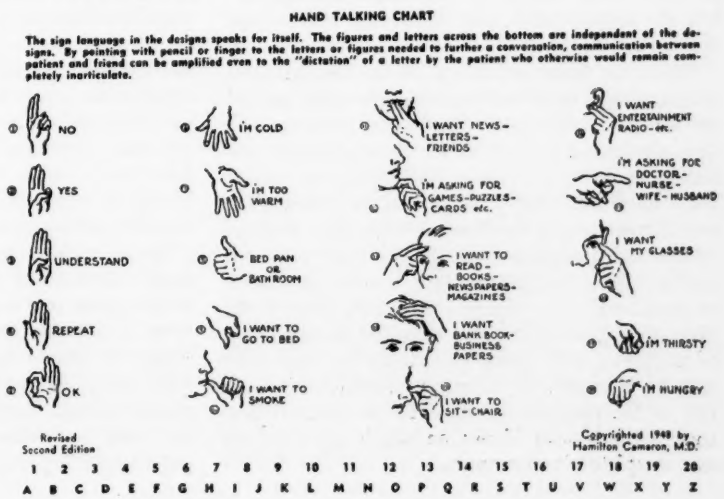
Dr. Cameron was stricken by coronary thrombosis in 1943, followed by cerebral embolism which resulted in right hemiplegia and "complete" aphasia. While still very ill, he realized that the one thing he needed most was to be able to communicate with those around him. With this admirably objective approach to a painful personal problem, he thought out and ultimately devised, with the help of Dr. Alfred A. Richman, medical director of the Manhattan General Hospital, New York City, the simple chart below.

Lest readers infer that Dr. Cameron's interest in solving problems is restricted to those with which he has had personal experience, it should be noted that his talents were directed toward aid for the unfortunate even before his own illness. After his graduation from Reliance Medical College, later merged with Loyola University in Chicago, in 1911, he began practice in the East. There he founded the Benevians, an organization which helped individuals advance in their chosen spheres of activity against odds. He was featured on the radio as the Benevian Visitor, philosopher to the sick, blind, and aged, calling on them at home and in the hospital.

Two and a half years after he was stricken, Dr. Cameron was able to express himself orally and have an artist make the drawings in the chart. He was given a three-year residency at Manhattan General Hospital for research on self-rehabilitation and for the development of techniques in this field.

The chart has been available to physicians and nurses in this country since 1945, and it is now distributed internationally in the 64 nations affiliated with the World Health Organization. Wall size charts are available to hospitals through the International Research Council, of which Dr. Cameron is founder and general director.

Physicians who wish more information may write Dr. Cameron at 601 West 110th Street, New York 25, New York.


HAND TALKING CHART
The sign language in the designs speaks for itself. The figures and letters across the bottom are independent of the designs. By pointing with pencil or finger to the letters or figures needed to further a conversation, communication between patient and friend can be amplified even to the "dictation" of a letter by the patient who otherwise would remain completely inarticulate.
NO
YES
UNDERSTAND
REPEAT
OK
I'M COLD
I'M TOO WARM
BED PAN OR BATH ROOM
I WANT TO GO TO BED
I WANT TO SMOKE
I WANT NEWS-LETTERS-FRIENDS
I'M ASKING FOR GAMES-PUZZLES-CARDS etc.
I WANT TO READ-BOOKS-NEWSPAPERS-MAGAZINES
I WANT BANK BOOK-BUSINESS PAPERS
I WANT TO SIT-CHAIR
I WANT ENTERTAINMENT RADIO-etc.
I'M ASKING FOR DOCTOR-NURSE-WIFE-HUSBAND
I WANT MY GLASSES
I'M THIRSTY
I'M HUNGRY
Revised Second Edition
Copyrighted 1948 by Hamilton Cameron, M.D.
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
A B C D E F G H I J K L M N O P Q R S T U V W X Y Z

Medical Provisions for Tax Returns

With the deadline for filing federal income tax returns extended to April 15 this year, few physicians are now concerning themselves with detailed analyses of provisions that are effective for the first time or changes that have been made in the law that formerly applied.

It is important, however, that all doctors have an understanding of the outline of the present tax structure, for it appears that more lay persons than usual, in preparing their returns, will assemble data on medical expenses. The law applying to income in 1954 is more generous than former laws in allowing medical deductions, so will therefore benefit a larger segment of the taxpaying public.

For some time elements within the medical profession have suggested that the cost of medical care could be reduced by altering tax laws to permit the deduction of every dollar spent for health purposes. This, they maintain, would also serve to combat propaganda issued by proponents of socialized medicine. There has been no indication that the suggestion will ever receive favorable action, but the changes now effective could possibly be construed as a step in that direction.

Formerly, no taxpayer could claim deductions for health expenditures unless the amount paid was in excess of 5 per cent of his adjusted gross income. This year all amounts exceeding 3 per cent of adjusted gross income are deductible. Allowable expenditures include amounts paid to doctors, dentists, nurses, and hospitals and payments for eyeglasses, artificial teeth, medical or surgical appliances, and premiums on health, accident, hospital, and medical insurance. Amounts paid for medicine and drugs, however, may be taken into account only to the extent that they exceed 1 per cent of adjusted gross income.

The new law also allows taxpayers to exclude from wages certain amounts received as wages for a period during which they were absent from work because of personal injuries or sickness. This amount may not exceed a weekly rate of $100. If the absence was due to illness, the exclusion does not apply to amounts received for the first seven days unless the taxpayer was hospitalized for at least one day during the period.

There has been no change in the law affecting deductions for blind persons. Partially blind persons are defined as those (1) whose central visual acuity does not exceed 20/200 in the better eye with correcting lenses or (2) whose visual field subtends an angle no greater than 20 degrees at its widest diameter. Physicians may be asked to certify such findings.

The few paragraphs above give only a general outline of tax provisions which may be of interest to physicians. The principles governing these provisions are extended in some instances to dependents of taxpayers, with special deductions for those more than 65 years of age. Complete information is available in the pamphlet supplied by the Internal Revenue Service—with blanks on which the physician may complete his own return.

Tribute to a Friend

Editor's Note. The following tribute to the late Dr. A. Morris Ginsberg, clinical professor of medicine at the University of Kansas School of Medicine, was written by Dr. E. Grey Dimond, chairman of the Department of Medicine at the school.

The University of Kansas School of Medicine, and particularly its Department of Medicine, lost a loyal friend and advocate in the death of Dr. A. Morris Ginsberg.

Dr. Ginsberg experienced his first coronary occlusion about six years ago. However, he returned to an active practice, continued his teaching of medical students, and just one week before his death was at the school helping give senior oral examinations. His final illness began in his office while he was examining a patient. What more fitting stage for a physician?

His association with the medical school began the year he opened his office. Through the years his kindly, sympathetic approach to the sick and his equally kind and courteous conduct with students left vivid memories among the graduates of this school. Dr. Ginsberg conducted himself in a gentle, humane manner, softening wisdom with understanding and humor.

Those of us who have come to the school in recent years found a warm welcome from Dr. Ginsberg. His long association with the school and with the community's medical practice gave him depth and perspective and made him a wise and valued counselor.

Although to all of us Dr. Ginsberg personified the art of medicine, early in his career he conducted problems in research, especially in the field of coronary flow. One paper published in *Archives of Internal Medicine* almost 20 years ago (55:42, 1935) is still a standard reference. In that paper Dr. Ginsberg reported his experiences while working with Dr. Stoland in using 50 per cent glucose by vein with a resulting significant sustained increase in coronary flow. Other fundamental studies carried out over a period of 17 years resulted in 31 contributions to scientific publications.

In recent years his gifts to the school permitted annual awards for scholastic achievement and the establishment of a student loan fund, which the school gratefully named in his honor.

Dr. A. Morris Ginsberg will not be forgotten. The school, its students, and its staff learned and gained through the years of association. Throughout our state and wherever our students have gone, physicians will pause and say, "He was a gentleman."

PR Programs for County Societies

A new publication of the American Medical Association, *County Medical Public Relations Manual,* is now being distributed by the A.M.A. to state medical societies and will soon be available to county groups. Prepared by the Department of Public Relations, the booklet is a comprehensive textbook on public relations outlined for most practical use at the county level.

Cancer Conference in March

Eight guest speakers who are recognized specialists in their fields will participate in the seventh annual Mid-West Cancer Conference, to be held at the Broadview Hotel, Wichita, on Thursday and Friday, March 24 and 25. The program outline includes scientific sessions on both days with a banquet for physicians and their wives on Thursday evening.

Biographical material about two of the speakers was published in the December issue of the JOURNAL. Three more sketches are presented here, and the remaining three will appear in the February issue.

One of the two pathologists who will take part in the conference is Dr. Elson B. Helwig, Washington, D.C. Dr. Helwig is a professorial lecturer in pathology at George Washington University. He is especially interested in dermal and gastrointestinal pathology, and in 1954 he was invited to give the 20th annual seminar of the American Society of Clinical Pathology, the subject being dermal pathology. He is a diplomate of the American Board of Pathology and a member of numerous professional organizations. During World War II he served as executive officer of the 18th Medical General Laboratory and as consultant in pathology in the Pacific Ocean areas.

Dr. George T. Pack of New York, a surgeon, will make his second appearance at a Mid-West Cancer Conference this year. He spoke at the 1951 sessions and was so well received that he was invited again in 1955. Dr. Pack is clinical professor of surgery at New York Medical College and associate clinical professor of surgery at Cornell Medical College. He is attending surgeon at Memorial Cancer Center, New York, and of the Pack Medical Group. He holds membership in a number of surgical specialty groups and in addition is a diplomate of the American Board of Radiology.

An oncologist who will speak is Dr. Herbert Nagler of Philadelphia. He now serves as associate in medicine (geriatrics) at Hahnemann Medical College, oncologist at Lucien Moss Home for Incurables, and associate at the Home for the Jewish Aged, Philadelphia. Although he received his preliminary degree in this country, he completed his medical work abroad and won his medical degree from the University of Basle, Switzerland, in 1935. During World War II he served as a flight surgeon.

In addition to the above, the following will take part in the program: Dr. Richard H. Chamberlain, Philadelphia; Dr. John R. McDonald, Rochester; Dr. Robert Dulaney Moreton, Fort Worth; Dr. C. C. Little, Bar Harbor, and Dr. Henry K. Ransom, Ann Arbor.

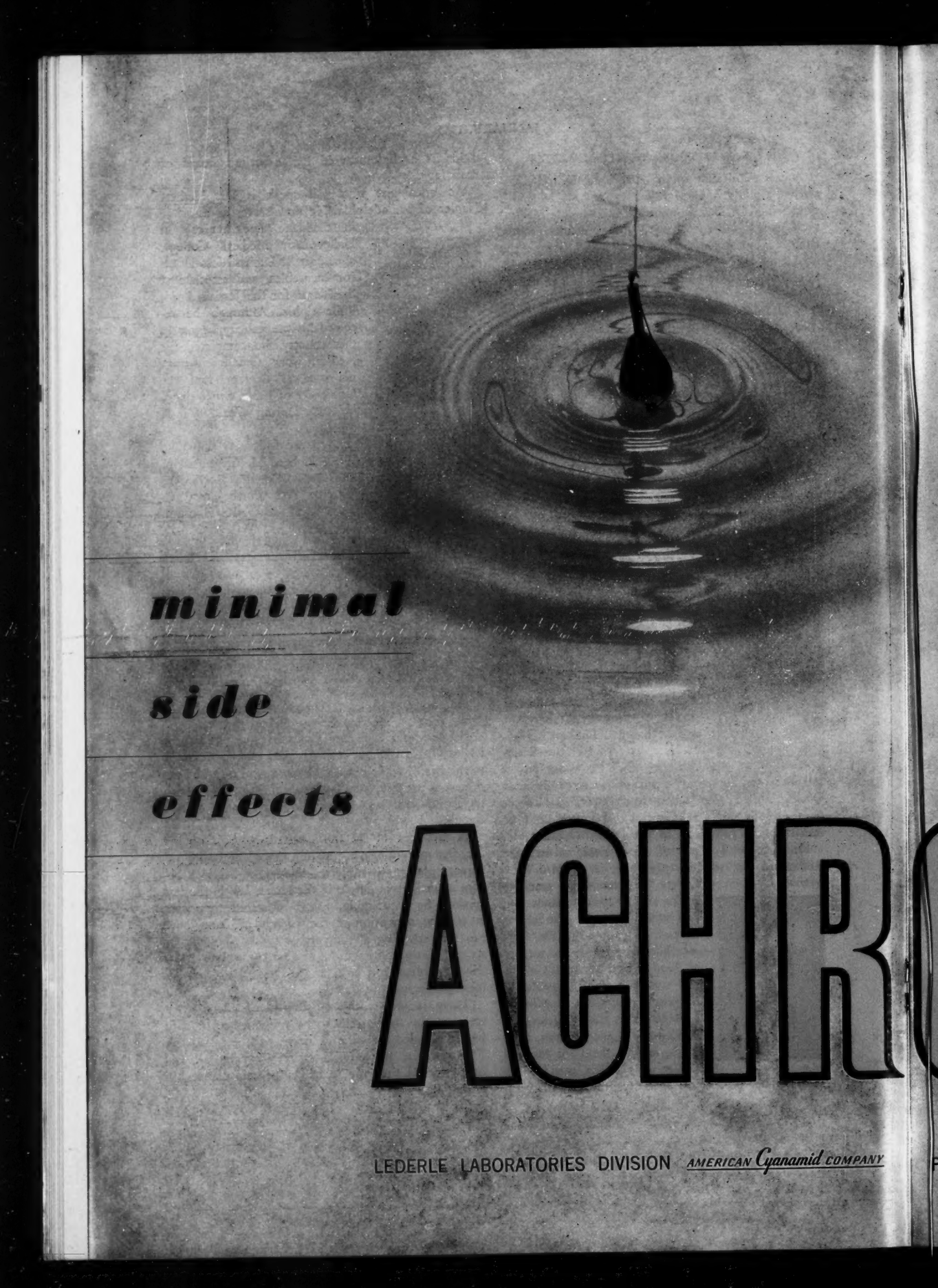
THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

Because this is a new Congress and under new leadership, a number of new bills can be expected in the health field. But the Democrats also can be expected to devote a vast amount of time to health legislation that was previewed last session by the Republicans.

In fact, one of the more prominent bills on the list, that providing federal reinsurance of health insurance plans, was subjected to lengthy hearings before it finally met defeat in the House late in the last session. So thoroughly was it dissected then that it will be surprising if the friends of reinsurance can find anything else favorable to say about it, or its critics can find anything else wrong with it. How this Republican bill will fare in Democratic committees now is one big question.

There is always the possibility, of course, that some of the major bills to be presented again will be so amended that new decisions will be called for. For example, the administration's experts all fall have worked tirelessly to make the reinsurance bill more palatable.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Like the reinsurance bill, the proposal to revamp the procedure for distributing public health grants to states was well worked over last session. It passed the House, but the Senate committee was unable to untangle all the knots it discovered, so there was no final action. This, too, is up again this year, labeled as difficult and touchy but nonpartisan.

Another well-advertised bill coming up for action is that to set up a program of contributory health insurance for federal employees. Last session a Senate committee held a one-day hearing on this bill, admittedly merely to get the proposition "on the record" so it could be freely discussed between Congresses. A task force from the Civil Service Commission has been trying to hammer out a more workable version of the bill, and has found the task a formidable one. But despite the complications, Congress will be asked to enact some bill of this type.

Although the bill definitely is of Republican origin, there is no reason to expect that it will receive a hostile reception from the Democrats in either House. It is generally accepted as a too-long delayed attempt to bring the federal government into line with private industry.

The bill for expanding medical care for military dependents has about the same history. After months of planning and conferences, bills were introduced last year in House and Senate to get the idea out into the open for the benefit of Congress and the public. Because the plan is so highly controversial, however, no hearings were held last session. The same bill is going before Congress again.

Here the fundamental issue is whether military hospitals and uniformed physicians shall supply the preponderance of this service to dependents, or the dependents shall be treated largely by civilian physicians and in civilian hospitals.

Last session the Defense Department prepared the draft of a bill to set up a number of military medical scholarships. Because bills originating in one department that might affect another first must be submitted to the latter for comment, this bill was turned over to Mrs. Hobby's Department of Health, Education, and Welfare. There it rested until after Congress adjourned. The 84th Congress will be asked to enact the bill, possibly as an alternative to extending the Doctor Draft, which is scheduled to expire next July 1.

Efforts will be made, but not necessarily with the Eisenhower administration's help, to enact some sort of legislation for federal guarantee of hospital mortgage loans. This subject was gone into in great detail last session by Mr. Wolverton's House Interstate and Foreign Commerce Committee, but the committee finally turned down Mr. Wolverton and refused to report out the bill for action. It had widespread labor support last year, but was opposed by the A.M.A. as discriminatory, in that it would offer more assistance to closed-panel practice than to other forms of medical practice.

Indications are that Mrs. Hobby's department will sponsor legislation to aid medical schools, a subject that was not taken up in the last Congress but that attracted considerable attention in years past.

House of Delegates, A.M.A.

At the mid-winter meeting of the American Medical Association at Miami, December 2-5, Kansas was represented by its senior delegate and by the president of The Kansas Medical Society, Dr. Murray C. Eddy. Dr. Eddy served as alternate because one of our regularly elected delegates, Dr. George Gsell, was precluded from attendance by the death of his mother-in-law.

The House of Delegates digested and acted upon between 50 and 60 reports and resolutions. Here are a few examples.

OSTEOPATHY

Negotiations are completed for "On Campus" examinations of five of six schools of osteopathy to determine the quality of training therein. Committees for doing this are being arranged, and it is hoped that an unbiased report will be available for the A.M.A. in Atlantic City next June.

MEDICAL ETHICS

The resolution presented by Kansas to delete Section Eight of Chapter One relative to ownership of drugstores from the code of ethics was rejected by the House, though the Reference Committee admitted some changes might be advisable. A change in another part will allow patents to be acquired by physicians on remedies, instruments, and appliances so long as profit therefrom does not retard research or restrict benefits to the public.

DOCTOR DRAFT LAW

The majority opinion was to the effect that the present Doctor Draft Law should not be extended beyond June 30, 1955, when it automatically expires. The Board of Trustees and the Council on National Defense are requested to continue their efforts to assure adequate medical care for the armed forces. A more permanent solution to the problem seems to be the development of a career Medical Corps with adequate compensation for medical graduates.

RE-INSURANCE PROGRAM

The beautiful and talented Mrs. Oveta Culp Hobby, Secretary of Health, Education, and Welfare,

extolled the virtues of reinsurance and received a polite applause for her efforts. In rebuttal, Mr. Edwin J. Faulkner, president of the Woodman Accident and Life Insurance Company of Lincoln, Nebraska, presented innumerable reasons against its acceptance. His very practical approach was given a rousing ovation. We were glad Mrs. Hobby remained to hear his remarks. We were sure she noted the different receptions of the two speeches.

NEW A.M.A. GERIATRICS UNIT

This body on a national level is to aid state and county societies as well as the public in a liaison capacity relative to pertinent information.

HEALTH FAIR

To this delegate one of the most important and worth-while events was a Health Fair put on by the county, state, and national medical associations for the public. There were 18 exhibits on such subjects as Accidents, Nutrition, Heart, Alcoholism, and diseases, particularly those related to so-called public health. Briefly this was unsurpassed as a public relations medium as is witnessed by the fact that 15,000 people visited the fair in one day. About half of the exhibits were loaned by the A.M.A. The others came from various organizations such as the American Cancer Society, etc. Kansas could have a similar program concurrently with our state meeting.

Dinner Key Auditorium adequately housed scientific and commercial exhibits, and adjoining it on one side were four spacious halls devoted to Internal Medicine, Surgery, Motion Pictures, and Color Television, respectively. While we delegates are always limited as to time spent in scientific meetings because of policy-making responsibilities in the House of Delegates, we did manage to visit briefly all departments. We are, therefore, certain that the 4,000 doctors in attendance received all they could absorb. There were about a dozen Kansas doctors in Miami for the meetings. This is a rather small representation. We hope there will be a larger group in Atlantic City next June.

Respectfully,

L. S. Nelson, M.D.
Senior Delegate

A new policy regarding immunization against influenza has been agreed upon by the Surgeons General of the Army, Navy, and Air Force. Accordingly, vaccine was administered to service men before a November 15 deadline, usually the time of the onset of the usual winter respiratory diseases.

BLUE SHIELD

Statistics released recently revealed that the 75 Blue Shield plans are making payments to doctors approaching $300 million annually. This represents an increase of 20 per cent over the preceding year.

These Blue Shield plans were able to return in form of benefits, on the average, 84.07 per cent of membership income with operating expenses being 11.23 per cent.

Private expenditures for hospital and medical care during this period were estimated at $9,866,000,000. Blue Cross, Blue Shield, commercial insurance, union plans, and all other types of coverage provided benefits totaling $1,919,200,000, according to the report.

Of the total private expenditures, 29 per cent went for physicians' services, 28.6 per cent for hospital care, 23 per cent for medicines and appliances and the balance for dental and miscellaneous services.

Total earned income for all carriers in the health care field totaled $2,404,600,000. This income, broken down by category of carrier, is as follows:

Blue Cross	$ 708,400,000	
Blue Shield	280,200,000	
Commercial Insurance	1,181,400,000	
Community Non-Profit Plans	56,300,000	
Union Health and Welfare Plans	81,700,000	
Employer-Employee Plans	49,600,000	
Prepayment Group Clinics	17,800,000	
Miscellaneous	30,000,000	(approx.)

For every dollar invested in health insurance, 80 cents was returned in the form of benefits. This ranged from 50 cents for individual contracts written by commercial carriers to 100 cents in the case of student health service. Blue Cross paid 88 per cent of premium income in benefits, Blue Shield 84 per cent.

The preceding statistical information is significant in that it points out the tremendous progress made by voluntary health plans and, at the same time, reminds us of the magnitude of needs not yet fulfilled.

Since only 20 per cent of total hospital and med-

REGULAR FEATURE OMITTED

Readers of the JOURNAL will note that one regular feature, ordinarily supplied by the University of Kansas School of Medicine, is omitted in this issue. The series which alternates Clinicopathological Conferences and Tumor Conferences is omitted now but will be resumed in the February issue of the JOURNAL.

ical care expenditures were covered by some form of insurance for 1953, it is apparent that many areas of need in the health care field are still untouched so far as prepayment is concerned. Some of these areas are home and office calls, care in nursing homes, home care for prolonged illness patients, diagnostic coverage, x-ray and laboratory services in doctors' offices, and out-patient treatment. Many of these needs are difficult to insure because of the tendency for overuse; however, there is a great deal of clamor from the public for comprehensive coverage.

Perhaps of most immediate importance to prepayment plans is the problem of finding ways to offer comprehensive coverage for services that are considered insurable. For example, many persons covered by insurance feel disappointed when they have surgery and hospital care and find that their coverage is only a partial payment. They have budgeted for insurance and then have to budget an additional amount at time of service to pay the total bill. In many instances this "additional payment" is a substantial amount.

Blue Shield has attempted to solve at least part of this problem with a "service benefit" provision for lower income groups. Unless the income limit for service benefit members is kept realistic, however, this provision tends to defeat its purpose.

Kansas Blue Shield and Blue Cross are studying ways for giving more comprehensive coverage to their members at a rate compatible to steady, sustained growth.

ACTIVITIES OF MEMBERS

A paper by Dr. Lewis L. Robbins, Topeka, "Emotional Reactions to Frustration and Failure," was published in the November issue of *American Practitioner and Digest of Treatment* as part of a feature section on "Stress Situations."

Dr. R. R. Melton, Marion, suffered the loss of his office, equipment, and records in a fire on December 1.

Dr. Simon E. Hershorn, Wichita, is now a diplomate of the American Board of Radiology.

Dr. William J. Reals, Wichita, addressed the medical technologists of Sedgwick County on the subject of "Protein-Bound Iodine Determination" on November 17.

Dr. H. R. Wagenblast has been granted a leave of absence from the Wichita-Sedgwick City-County Health Department for a year of postgraduate study at the School of Public Health, University of California.

Dr. J. Robert Twinem, who has been practicing in Garden City for three years, is now associated in practice with Dr. Edmer Beebe in Olathe. Dr. Twinem is a graduate of the University of Colorado School of Medicine.

Dr. C. C. Nesselrode, Kansas City, was recently elected an honorary member of the board of the American Cancer Society.

Dr. Hansel Benvenuti, who resigned as medical director of the Sedgwick County Hospital, Wichita, on December 1, was replaced in the Wichita position by Dr. Verl J. Throckmorton, who recently completed a surgical residency at Wesley Hospital there.

The Hertzler Clinic, Halstead, announces that Dr. R. G. Holt, who was recently released from service with the Navy, is now a member of its staff.

Dr. James A. McClure recently completed 21 months of service with the Navy and has returned to his urological practice in Topeka. While in the Navy he served on Atlantic transport duty, at Corpus Christi, Texas, and at Bremerton, Washington.

Dr. C. J. Kurth, Wichita, spoke on "Business and Mental Health" before the Town Club of Business and Professional Women in Wichita last month.

Dr. Robert E. Riederer, Olathe, has been appointed physician at the Kansas School for the Deaf, replacing Dr. Houghton S. Albaugh, who resigned after 15 years in the position.

Dr. Frederick A. Gans, Salina, was speaker at a recent meeting of the 4-H Club of Salina School District 3. He spoke on health and safety subjects.

Dr. Newman V. Treger, Topeka, represented the medical profession in a recent program on vocational guidance for students at Topeka High School.

Dr. Charles V. Minnick, Junction City, announces that Dr. Murray F. Minthorn is now associated with him in practice. Dr. Minthorn, a graduate of the University of Nebraska College of Medicine, has just completed a tour of duty with the Air Force.

Dr. Howard E. Snyder, Winfield, was recently named a director of the American Cancer Society.

Dr. M. H. Waldorf, Jr., expects to be released from military service this month and will return to his practice in Greensburg. Dr. J. R. Bradley, who has been practicing in Greensburg, has received orders to report for service with the Air Force on February 21.

Dr. Ray T. Parmley, Wichita, was recently named a director of the American Society of Anesthesiologists.

Dr. W. Clarke Wescoe, dean of the University of Kansas School of Medicine, was one of the participants in a panel discussion held in Kansas City for the benefit of 84 physicians from foreign countries who are now taking training at Kansas City hospitals.

Dr. Richard B. Williams, formerly of Pratt, opened an office in Independence last month.

Dr. David H. Rau, Lyons, has been called to a two-year tour of duty with the Army. He was ordered to report at Fort Sam Houston, Texas, on January 2.

Dr. Homer L. Hiebert, Topeka, took part in a panel discussion on "Fundamental Problems in the Clinical Use of Isotopes" at the annual meeting of the Radiological Society of North America, Inc., held in Los Angeles, December 6-11. Dr. Frank E. Hoecker, professor of physics at the University of Kansas, conducted a refresher course on "Recent Advances in Clinical Radioisotope Technics," at the same meeting.

Dr. Mahlon Delp, professor of medicine at the University of Kansas School of Medicine, has been elected to membership in the American Clinical and Climatological Association. The group, founded in 1884, is restricted in membership to 175 doctors, with only five new members elected each year.

The Ball Clinic, Manhattan, announces that Dr. Howard M. Lamborn, Jr., has become a member of its staff. Dr. Lamborn, who practices obstetrics and gynecology, was graduated from the University of Kansas School of Medicine in 1942, served three years in the Navy, completed a three-year residency in Cincinnati, and has recently been practicing in California.

Dr. H. Alden Flanders has returned to practice with the Eddy Clinic, Hays, after spending 21 months in the Navy. His period of service was spent in the Pacific, aboard a transport during the Korean conflict and aboard a ship evacuating refugees from Indo-China in recent months.

DEATH NOTICES

Z. Hosea Snyder, M.D.

Dr. Z. H. Snyder, 84, an honorary member of the Washington County Medical Society, died at St. Anthony's Hospital, Hays, on December 9. He had been living in Hays since he retired from practice last summer, but before that time he had lived in Greenleaf and had practiced there more than 50 years. He was a graduate of the Keokuk (Iowa) Medical College.

Richard Clark Lowman, M.D.

Dr. R. C. Lowman, 87, who had practiced medicine more than 60 years, died on December 20. An honorary member of the Wyandotte County Society, he was feted in 1951 as the oldest practicing physician in the area. He was graduated from Kansas City Medical College in 1890, and he began practice in Kansas City in 1901, specializing in surgery. He was a fellow of the American College of Surgeons.

Herbert Melville Webb, M.D.

Dr. H. M. Webb, 77, an honorary member of the Allen County Society, died December 16 at the Veterans Administration Hospital, Kansas City, where he had been a patient since July. He received his medical education at Lincoln (Nebraska) Medical College, Eclectic, graduating in 1906. He immediately opened an office in Humboldt and continued to practice there until his retirement.

The following are new officers of the Wyandotte Academy of General Practice: president, Dr. Matthew R. Fitzpatrick; vice-president, Dr. Karl M. Rottluff; secretary, Dr. William D. Hawley, and treasurer, Dr. James B. Pretz.

Dr. Thomas F. Morrow, Wichita, who also serves as psychiatrist at Prairie View Hospital, Newton, spoke on "Elements of a Therapeutic Community" at the annual meeting of Meadowlark Homestead, Newton, last month.

Dr. John D. Hilliard, Medicine Lodge, was recently named coroner of Barber County.

The following physicians will serve as officers of the Topeka Blood Bank during 1955: president, Dr. Joseph E. Gootee; secretary, Dr. Robert H. O'Neil; board members, Dr. O'Neil, Dr. William H. Crouch, Dr. A. A. Fink, Dr. D. B. Foster, Dr. John W. Cavanaugh, and Dr. David E. Gray.

COUNTY SOCIETIES

Dr. Albert N. Lemoine, Jr., of the University of Kansas Medical Center, was guest speaker at a meeting of the Sedgwick County Society held in Wichita on December 7. His subject was "Strabismus in Childhood."

Members of the Auxiliary in Sedgwick County entertained their husbands and guests at a Christmas party and dance on December 15.

Dr. Herschel R. Turner, Hope, was host to members of the Dickinson County Society at a turkey dinner at the Legion Hall at Hope on November 18.

Dr. Kenneth J. Gleason, Independence, was elected president of the Montgomery County Society at a meeting held the latter part of the year. Dr. Hubert C. Martin, Coffeyville, was named vice-president, and two physicians from Independence, Dr. William G. Chappuie and Dr. Gerald C. Bates became secretary and treasurer.

A feature of the 53rd annual dinner meeting of the Shawnee County Society, held at the Jayhawk Hotel, Topeka, on December 6, was the presentation of membership pins in the 85-50 Club to Dr. John A. Crabb and Dr. Elvenor A. Ernest. The club is composed of physicians who have attained the age of 85 or have completed 50 years of practice. Those who have become members in former years are Doctors William L. Borst, Michael A. Floersch, Ralph L. Funk, William H. Greider, Henry B. Hogeboom, Clarence H. Kinnaman, Charles H. Lerrigo, James H. McNaughton, John F. Northrup, Joseph C. Shaw, William L. Warriner, Charles L. Youngman, and George W. B. Beverly.

Speaker of the evening was Mr. Dave Livingston, Washington, Iowa. A short talk was also given by another guest, Mr. Basil O'Connor, president of the National Foundation for Infantile Paralysis.

The new officers of the group, to serve in 1955, are: Dr. D. Bernard Foster, president; Dr. Clyde B. Trees, president-elect; Dr. Charles S. Joss, vice-president; Dr. Willis L. Beller, secretary, and Dr. B. I. Krehbiel, treasurer. Dr. Howard E. Roberts was named to the board of directors, Dr. Byron J. Ashley to the board of censors, and Doctors William H. Crouch, David E. Gray, and Walter Mau to the medical service board.

A meeting of the Ford County Society was held at the Lora-Locke Hotel, Dodge City, on December 14. Dr. A. A. Fink, guest speaker from Topeka, discussed "Early Diagnosis of Cancer."

Dr. Eugene J. Bribach was elected president of the Atchison County Society at a meeting held at the home of Dr. Wayne O. Wallace, Atchison, on December 7. Dr. Francis I. Stuart was named vice-president, and Dr. Ira R. Morrison was chosen as secretary-treasurer. Other officers are censors elected for three-year, two-year, and one-year terms, Dr. Frank K. Bosse, Dr. G. A. Patton, and Dr. Wallace.

Dr. Royal A. Barker, Peabody, presented a paper on the causes and treatment of hematuria at a meeting of the Marion County Society held on December 7. The following officers for 1955 were elected: president, Dr. Herman F. Janzen; vice-president, Dr. Peter D. Enns, and secretary-treasurer, Dr. Wendell M. Tate.

A meeting of the Clay County Society was held at the Clay Center Hospital on December 8. Dr. William H. Crouch, Topeka, guest speaker, discussed erythroblastosis. At the business session the following officers were elected: president, Dr. Carl H. Ruff;

vice-president, Dr. Roy B. McVay; secretary-treasurer, Dr. F. C. Shepard. The same officers will serve for the hospital staff.

Members of the Wyandotte County Society met at the City-County Health Building, Kansas City, on December 21. Dr. William W. Abrams presented a paper, "Medical Preparedness for Civil Defense." A business session followed.

Dr. Paul Schraer was elected president of the Cloud County Society at a meeting held at Concordia on December 14. Dr. N. C. McCubbin was named vice-president, and Dr. Lindell C. Owensby became secretary-treasurer.

Heart Research at K.U.

A new research project—in which an investigator will delve into the functions of the human heart—has been started at the University of Kansas Medical Center through the newly established McIlvain Biochemical Cardiovascular Laboratories, occupying five rooms on the fourth floor of the Medical Sciences Building.

Dr. Santiago Griscolia, who holds the "Established Investigator" award of the American Heart Association, will conduct the study. It will concern the chemical processes by which the heart muscle works normally as well as the enzyme, hormone, and electrolyte changes in heart disease.

The McIlvain trust was established in 1950 by the late Mrs. Frederic Ervine McIlvain, and a grant of $54,000 was made to the medical center last April.

An additional grant of $2,000 was awarded the laboratory last month by the Kansas Heart Association. This is in addition to the association's contribution to research on a national basis, 12 cents of every dollar received.

Attend A.M.A. Meeting

The following physicians from Kansas attended the American Medical Association clinical session at Miami last month: Doctors Henry S. Blake, W. M. Delaney, Dwight Lawson, Chester E. Joss, Manuel E. Pusitz, Clarence K. Schaffer, and Rudolph Chess of Topeka; Farris D. Evans and George L. Thorpe of Wichita; Loren E. Dickelmann, Hutchinson; Murray C. Eddy, Hays; Walter M. Furst, Norton; Ferdinand C. Helwig, Kansas City; L. S. Nelson, Salina; Marvin O. Steffen, Great Bend, and C. C. Underwood, Emporia.

Medical School Enrollments

A survey of applicants for medical training was made recently by the Association of American Medical Colleges and showed that availability of medical schools and a high per capita income in a given area have a bearing on the number who desire to enter the profession.

Various geographical areas in the country have about the same number of freshman places available in medical schools, with five per 100,000 population reported. The larger the number of practicing physicians in a state relative to population, the greater the interest in obtaining medical education, it was found. Number of colleges in the state is not necessarily related to the volume of the applications, nor is the per capita income of physicians a consideration.

For the country as a whole there has been an overall decrease in the number of applicants. Areawise, the Middle Atlantic states lead with 14 applicants per 100,000 population. The national average is nine. The District of Columbia is first with 18 per 100,000, Nebraska second with 17, and Utah and New York third with 15.

Two-thirds of all freshman medical students enter schools in their own states, but many places are filled by out-of-state residents.

Television in Medical Education

A program on "The Potential Use of Television in Postgraduate Medical Education" will be presented at the Palmer House, Chicago, on February 5 under the sponsorship of the A.M.A. Council on Education and Hospitals. This is the first of a series of "workshop" type conferences on one particular aspect of postgraduate education.

Television is the subject of the first meeting because of the extreme interest shown in this medium recently. The program is planned to present both the educational and technical aspects of the subject so that hospital and specialty society representatives at the meeting will be able to obtain a broad picture of the medium and determine whether or not it is something they might use in their own programs.

The morning session will be devoted to considertions of the purely educational aspects of television. The afternoon session will deal with technical considerations and financing. Participants will be drawn from the fields of general education, television, industry, medicine, medical education, pertinent government agencies, and others. Demonstrations will be given showing actual camera chains and receiving equipment. The group will also visit a local television station in action.

Peripheral Embolic Arterial Occlusion

Eugene L. Petry, M.D.
Wichita, Kansas

Peripheral arterial embolism is not an uncommon medical and surgical emergency. Most often it is seen in patients with underlying cardiac disease, patients whose condition already is precarious. Thus the episode of embolism poses itself as an additional hazard to a severely ill individual. Unless diagnosis is prompt, unless treatment is adequate, the prognosis for life and limb is often grave.

ETIOLOGY

In a patient presenting signs of acute arterial insufficiency, the diagnosis of embolism depends upon demonstrating a source of thrombosis. Usually there will be a past history of cardiac disease, but the cardiac pathology may remain unsuspected until the occurrence of the embolic episode.[1] In series reported by various authors, the heart has been considered the site of primary thrombosis in from 60 to 96 per cent of cases.[2, 3] Pratt[4] reports that 80 per cent of peripheral emboli are associated with left sided heart disease and that 25 per cent of patients dying of heart disease have embolic episodes.

The cardiac pathologies encountered include rheumatic heart disease, myocardial infarction, arteriosclerotic heart disease, subacute bacterial endocarditis, thyrocardiac disease, and Cooley's anemia. It is not uncommon for surgical procedures on the heart to dislodge fragments of thrombus into the peripheral circulation.[5]

Extracardiac sources of thrombosis occur in pulmonary phlebitis, aortic and arterial aneurysms, or on calcific plaques in sclerotic arteries. Paradoxical emboli arising from sites of thrombosis in the systemic veins may gain access to the arteries of the extremities via interatrial septal defects. Groth[6] has reported the embolic occlusion of the common femoral artery by tumor cells migrating from the pulmonary metastases of a sarcoma primary in the right tibia.

In those patients with cardiac disease, the historian may often develop the account of recent changes in the cardiac status:[7] the onset of or changes in the severity of congestive failure; changes in rhythm; changes in digitalis dosage. Reynolds and Jirka state that embolism may occur with physical exhaustion;[7] but Sprague and Westinghouse, in a review of 75 patients suffering 77 attacks of acute arterial occlusion, noted only 5 episodes occurring during or after unusual exertion, whereas 35 occurred during rest in a bed or chair.[8] Operative procedure, the use of antibiotics, and digitalis itself may be additional predisposing factors to thrombosis.

The relative incidence of these varied etiologic conditions in arterial embolism may be observed in Haimovici's analysis of 330 emboli occurring in 228 patients.[3]

TABLE I

ETIOLOGY OF EMBOLISM IN 228 PATIENTS (HAIMOVICI)

Rheumatic heart disease	40.4%
Myocardial infarction	31.5%
Arteriosclerotic heart disease	19.3%
Subacute bacterial endocarditis	4.8%
Pulmonary venous thrombosis	0.9%
Undetermined	3.1%

Rheumatic heart disease, the most common cause of embolism, warrants further discussion. Haimovici found it to be the etiology of 40.4 per cent of peripheral emboli; it accounted for 64 per cent of the emboli in Warren and Linton's series.[2] Approximately 5 per cent of patients with this condition will have emboli, and of these 60 per cent will have multiple episodes of occlusion.[9] In a necropsy study of 178 rheumatic hearts, Graef[10] found the incidence of auricular thrombosis to be 13 per cent.

The factors predisposing to thrombosis in a rheumatic heart are stated by Hay and Levine[11] to include the duration of disease, the persistence of rheumatic activity, the presence and duration of failure, the presence of mitral stenosis, the presence of auricular fibrillation, and the age of the patient. Their statistics show that thrombosis occurs three to four times as often in fibrillators as compared to patients with a normal rhythm. In another necropsy series it was found that 90 per cent of persistent fibrillators develop intra-auricular thrombi.[12] Haimovici reported the following incidence of cardiac defects in his patients with rheumatic hearts:

Auricular fibrillation	81.5%
Mitral stenosis	79.3%
Mitral regurgitation	55.4%
Aortic regurgitation	23.9%
Aortic stenosis	6.5%

Daley's group[13] reported the incidence of fibrilla-

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Dr. Petry is now interning at Wesley Hospital, Wichita, Kansas.

tion and mitral disease to be 90 per cent and 97 per cent respectively in 194 patients with rheumatic hearts and embolic episodes.

Myocardial infarction is not uncommonly complicated by embolism. It has been reported that thrombo-embolic episodes occur in 15 to 25 per cent of those cases which do not receive prophylactic anticoagulants.[14, 15, 16] It is probable that the incidence of thrombosis and subsequent embolism is related to the extent of necrosis, the presence of shock, and the development of arrhythmias. Gilchrist and Tulloch[17] have noted an increase in the plasma fibrinogen following infarction, the titer reaching a peak in about one week. They relate the extent of elevation to the severity of infarction. In cases of silent infarction, the symptoms of embolism may be the first warning of danger and warrant a complete cardiac evaluation.

SITES OF OCCLUSION

Intravascular thrombi may embolize to any part of the arterial tree. The many factors determining the final resting place of the clot are not fully understood. Of importance are the size of the clot, the size of the vessels, and the force of the blood stream which carries the embolus. Occlusion almost always occurs at the bifurcation of arteries where the diameter of the vessel is markedly diminished. The higher incidence of occlusion in the lower extremities is explained, perhaps, by the lack of sharp turns for the embolus to encounter on its course. As is shown in Table II, the frequency of occlusion in the lower limbs is three to four times that in the upper.

TABLE II

COMPARATIVE FREQUENCY OF EMBOLISM TO THE ARMS AND LEGS

	Haimovici (330 emboli)	*Warren & Linton (110 emboli)*
Arms	16%	21.8%
Legs	75%	64.7%
Aorta	9%	13.6%

The site of occlusion is of specific importance in predicting the prognosis of the extremity, though one cannot ignore such important factors as the presence of local vasular disease, the duration of occlusion, and the extent of secondary thrombosis. The results following occlusion in the upper extremities are consistently better than in the legs. Similarly, gangrene is much less likely to develop if the obstruction involves small distal vessels rather than large, proximal mainstem arteries. Thus necrosis invariably follows embolic occlusion at the aortic bifurcation. The sites of occlusion found by Haimovici[3] in 330 episodes of embolism are shown in Table III.

In Haimovici's series peripheral emboli accounted for 74.3 per cent of all embolic episodes in 228 patients. The incidence of peripheral embolization was 63.9 per cent in the group reported by Warren and Linton.

TABLE III

SITES OF OCCLUSION BY 330 EMBOLI (HAIMOVICI 1950)

Aortic bifurcation	9.1%
Common iliac	13.6%
External iliac	3.0%
Femoral	38.5%
Popliteal	14.2%
Posterior tibial	2.8%
Anterior tibial	2.8%
Axillary	4.5%
Brachial	9.1%
Radial	1.2%
Ulnar	1.2%

CLINICAL PICTURE AND DIAGNOSIS

The signs and symptoms of peripheral embolism are those of acute arterial insufficiency, the severity of symptoms varying with the degree of ischemia. The onset may be sudden or progressive. The initial symptom may be severe pain, but pain may not be apparent for several hours. The patient usually notes numbness, coldness, paresthesias, weakness, and paralysis. Examination of the limb reveals pallor and cyanosis, collapse of the superficial veins, lowered skin temperature, absence of the arterial pulses, hyporeflexia, hypesthesia, muscular weakness, and extreme muscular tenderness.

The sudden onset of severe pain traditionally has been considered the characteristic symptom of embolism. Some authors feel this to be so typical as to permit distinguishing embolism from occlusion by primary thrombosis. If one waits for such classical identification, many cases of embolism will remain undiagnosed. Saland[18] was unable to distinguish between embolism and thrombosis on the basis of symptoms alone. Sudden severe pain is not a constant finding, being present in only 50 to 65 per cent of cases.[2, 3, 18, 19] Haimovici noted sudden onset of pain in 50.5 per cent of cases, while in nearly 20 per cent the lesion was silent or the onset progressive.

TABLE IV

ONSET OF SYMPTOMS IN PERIPHERAL ARTERIAL EMBOLISM (HAIMOVICI 1950)

Sudden onset of symptoms	81.5%
Sudden pain	59.5%
Sudden numbness & coldness	21.7%
Progressive onset	11.7%
Silent occlusion	5.7%

The pain of embolism has been ascribed to vasospasm, impact of the embolus on the arterial wall, and to muscular ischemia. Lewis[20] denies that the instant of embolism is marked by pain. Reynolds and Jirka[7] note that the site of impaction becomes tender only after inflammation has begun. Lewis believes

that the tissue in which pain originates is muscle, and that the onset and severity of pain are related to the activity and subsequent ischemia of the part. Thus a person resting quietly in bed may note pain only after several hours, but he will have progressive numbness, weakness, and coldness beginning about 30 minutes after the occlusion. On the other hand, the rapid increase of metabolites associated with activity will produce severe, rapidly progressive pain before the neurovascular symptoms are evident. It is thus important for the historian to note the time of onset, the progression of symptoms, and the activity of the part concerned at the time of occlusion.

The loss of arterial pulsations is not a reliable guide in determining the level of occlusion. Severe vasospasm following a distal occlusion may obliterate the proximal pulses; at other times pulsation may be felt several centimeters distal to the obstruction due to transmission of pulsations through the clot itself (Nordentoft's sign).[21] Oscillometric studies are a valuable adjunct to digital palpation of the arteries and provide a more reliable method for determining the level of occlusion. Classically there may be found three oscillometric zones: a proximal zone of normal pulsation, a distal zone of absent pulsation, and an intermediate zone of diminished pulsation, the embolus lying near the proximal end of the intermediate zone.[21] If an oscillometer is not available, one may use in its place a sphygmomanometer inflated a little above the diastolic pressure.[22]

Examination of the extremity usually reveals a line of demarcation between the normal and ischemic tissue. If collateral circulation has developed, this line of demarcation may lie several centimeters distal to the level of obstruction. If the collaterals are markedly spastic, the line of ischemia may lie proximal to the embolus. The histamine flare test is considered by de Takats[23] as a useful means of determining the collateral supply. If erythema and a wheal fail to develop after the intradermal injection of histamine, the circulation is probably not adequate to preserve viability.

The diagnosis of embolic occlusion depends upon demonstrating a source of thrombosis in a patient suffering the syndrome of acute peripheral ischemia. At times, the source of thrombosis will remain unsuspected until suggested by the embolic episode. Embolism must be differentiated from other conditions which may cause acute ischemia: primary arterial thrombosis, acute thrombophlebitis, and thromboangiitis obliterans. If there is doubt as to the cause of ischemia, it is better to treat the case as embolism than to permit an undiagnosed embolus to condemn an extremity to gangrene or persistent arterial insufficiency.

Primary arterial thrombosis produces the same clinical picture as embolism but should be suspected when one obtains a past history of prior vascular disease with symptoms of intermittent claudication, trophic changes in the skin and nails, and coldness of the distal parts. Acute thrombophlebitis typically does not produce ischemia, though in some instances the arterial spasm may be so severe as to obliterate the pulses and produce gangrene.[24] In such cases the spasm will be relieved by sympathetic blocks which permit prompt return of color, warmth, and sensation to the part. Patients with thromboangiitis obliterans will usually have a prior history of migratory superficial thrombophlebitis.

PATHOPHYSIOLOGY OF ARTERIAL EMBOLISM

The event of embolic occlusion of a major arterial channel is the first stage of a series of events which further tend to embarrass the circulation to the extremity. These factors are considered by Haimovici[3] to be mechanical obstruction, vasospasm, secondary arterial thrombosis, and histiologic changes in the arterial wall.

The mechanical factor of embolism is complete arterial obstruction by the impacted clot with cessation of blood flow through the vessel. The region of ischemia resulting from such occlusion is smaller if distal vessels are obstructed than if the occlusion occurs in large mainstem arteries. When collateral pathways are present, the circulation to the tissues may remain sufficient to preserve viability unless these bypasses are secondarily obliterated by spasm or thrombosis. A friable embolus on striking the bifurcation of a vessel may shatter into many fragments which produce multiple occlusions of smaller vessels.[25] A similar problem may be present when multiple embolic episodes involve the same extremity.

Vasospasm involving both arterial and venous channels generally accompanies mainstem arterial occlusion. The extent and severity of spasm varies from case to case, assuming little or great importance. Myogenic spasm induced by direct trauma to the arterial wall at the site of impaction produces constriction only in the vicinity of the clot. It has been postulated that spasm of this origin prevents further distal migration of the embolus.[7] Neurogenic or reflex vasospasm arising from local irritation of the arterial endothelium may produce extensive obstruction of the arterial and venous channels throughout the extremity.[26] This spasm may involve the entire collateral system, producing a complete cessation of blood flow to the distal parts. The vessels proximal to the embolus may be so markedly constricted as to produce loss of pulsations and a level of ischemia several centimeters proximal to the original site of occlusion. In rare cases the arteries of the opposite limb may be involved, producing bilateral ischemia with unilateral embolism.[27]

Shortly after embolism occurs—perhaps as soon

as two hours—secondary thrombosis begins to develop in the arterial tree. The earliest clots form in the areas of greatest stagnation and injury, usually in the main arterial trunk immediately distal to the embolus. It is in this region that the stagnant column of blood is exposed to increased amounts of thromboplastic material liberated by the damaged arterial wall.[7] Iselin and de Balsac[28] postulate that clotting is related to altered physiology of heparin-producing cells in the arterial sheath. If the collateral circulation remains unimpaired, the distally propagating thrombus will extend only to that point where stagnation is relieved by blood re-entering the occluded artery. In cases where vasospasm produces stagnation along the entire length of the vessel, thrombosis will extend distally into the terminal branches. Thrombosis similarly proceeds in the collateral vessels when the rate of blood flow within them is reduced by spasm. Thus within a few hours the entire arterial system of the extremity may be obliterated by the process of post-embolic thrombosis.

With drastic reduction of arterial blood flow, it is expected that stagnation will likewise occur in the venous circulation of the limb. Laufman[29] observed minute thrombi forming in the sluggish venous blood stream following arterial occlusion. From these minute thrombi develops the segmental venous thrombosis which not infrequently occurs after acute arterial occlusion.

Within several hours after embolism, inflammatory reactions begin in the arterial wall adjacent to the clot. This reaction proceeds more rapidly in adventitia than in the medial and intimal layers.[3] Haimovici feels that this early arteritis is associated with a periarteritis which extends to adjacent veins producing phlebitis by direct extension. After a further lapse of time, the processes of repair or fibroplasia bind the embolus tightly to the arterial wall by fibrous tissue.

TREATMENT OF ARTERIAL EMBOLISM

As a medical and surgical emergency, peripheral arterial embolism demands prompt, adequate treatment. Within the period of a few hours, the physician must attempt a solution to the problem in order to obtain favorable results. All workers in the field agree that treatment must begin as soon as the diagnosis is made, but there is no agreement as to what comprises proper treatment of a limb affected by embolism. No particular regime of management gives consistently good results; therefore, each has its respective proponents and opponents.

The general aims of therapy are to preserve the life of the patient and to preserve the integrity of the limb. As has been mentioned, the cardiac status of patients with embolism is often precarious. It is beyond the scope of the present discussion to describe the management of cardiac conditions. The survival of the extremity affected by acute ischemia depends upon: (1) preserving and restoring the blood supply, (2) protection from physical and chemical trauma, and (3) preventing infection.

Efforts to preserve and restore blood supply are directed toward: (1) the relief of vasospasm, (2) the prevention of thrombosis, (3) surgical removal of the obstruction, and (4) the use of mechanical means to improve circulation. Ischemic tissue has little resistance to the deleterious effects of chemical and physical agents which tend to damage the skin and underlying tissues. Therefore, it is essential that in cases of embolism the limb be protected from pressure, abrasion, harsh chemicals, heat, or cold. Infection is usually not a serious problem in the early management of arterial embolism, but precautions should be taken to prevent bacteria gaining access to the devitalized tissues.

The methods of management of peripheral arterial embolism will be discussed under the following headings:

General procedures
Vasodilator drugs
Anesthetic blocks
Anticoagulants
Mechanical methods to improve the circulation
Surgical procedures
Enzymatic agents

General Procedures. In the management of peripheral embolism, certain general procedures serve as valuable adjuncts to the more specific modes of therapy. The serious nature of embolism, of course, warrants the patient being put to bed where the care of his limb may be rigidly supervised. As elevation of the part further diminishes the arterial supply, the limb should be placed in a dependent position. This may be accomplished by raising the head of the bed on shock blocks. Protective wrappings of cotton or sheet wadding protect the damaged skin from abrasions, and the danger of pressure may be obviated by placing the bed clothes over a protective cradle. Heat to the extremity is strictly contraindicated as it increases the rate of metabolic activity and, thus, hastens necrosis and gangrene. Though direct heat has been considered a means of producing dilatation of the spastic vessels, its use for this purpose is unwarranted and dangerous.

O'Leary et al.[30] suggest that reflex dilatation may be achieved by heating the uninvolved extremities. Their program of management includes heat applied in this manner for 30 minutes every 4 hours. Reynolds and Jirka[7] discuss the possibility that refrigeration may be used to retard the metabolic activities of the ischemic limb, thereby reducing the nutritional requirements of the tissue. Though refrigeration may offer possibilities, it is now most generally accepted

that it is best to maintain the limb at normal room temperatures.

Vasodilator Drugs. Vasodilator drugs have long been recommended for the management of arterial embolism. Papaverine, one of the earlier agents available for the relief of vascular spasm, is still one of the more popular agents.[4] Its advocates suggest that the drug be given in doses of half a grain every 4 hours and continued over a period of several days.[25, 27] However, McClure and Harkins[21] remark that papaverine is probably of little value in arterial embolism. Warren and Linton[2] similarly conclude that the drug is not an effective vasodilator. Massel[1] believes that papaverine hydrochloride produces little increase in the peripheral circulation after embolism and suggests that priscoline is the drug of choice.

Cooper et al.[31] evaluated the effectiveness of tetraethylammonium chloride in relieving acute arterial insufficiency in animals from which were resected the aortic bifurcation with the common iliac and common hypogastric arteries. The untreated control group died within 24 hours, but 70 per cent of those receiving TEAC survived with a return of function to the hind limbs within 2 to 6 days. These workers, therefore, consider TEAC to be a potent agent for the relief of arterial spasm.

Though the relief of vasospasm is indicated in the treatment of embolism, there are some who feel the hypotensive response to the generalized vasodilatation induced by potent vasodilatory drugs impairs further the already inadequate blood flow through the limb. Drugs acting only on the wall of the spastic arteries would seem to be of small avail in acute ischemia, since the circulation is inadequate to carry the agent to its target organ. The ganglionic-blocking agents such as TEAC produce such a generalized response as to be potentially dangerous.

Anesthetic Blocks. Another means of attacking the arterial spasm incident to embolism is by anesthetic blocks of the sympathetic ganglion or by spinal anesthesia. The use of these methods is generally accepted as an effective means of preventing or of reversing the reflex component of post-embolic vasospasm,[2, 4, 21, 30, 32] though the myogenic element remains unaffected. Reynolds and Jirka[7] believe that vasospasm aids in preventing further distal migration of the embolus into segments of the artery inaccessible to surgery.

The response obtained from anesthetic blocks is dependent on several interrelated factors: (1) the duration of occlusion, (2) the severity of spasm, and (3) the extent of secondary thrombosis. Use of blocks in the early hours of occlusion before secondary changes have developed permits a more favorable response. With severe uncomplicated vasospasm, blocks may produce a dramatic return of warmth and color to the ischemic limb. When secondary thrombosis has obstructed the collateral channels as well as the main arterial trunk, paralysis of the constrictor reflexes promotes no significant increase in blood flow. The response to anesthetic blocks is judged by observing changes in the degree of ischemia, a good response producing return of color, increased warmth, relief of pain and, sometimes, return of pulsations. At times the line of demarcation may move distally below the actual site of impaction as the proximal vasospasm is reduced.

Sympathetic blocks are performed by injecting anesthetic agent into the appropriate sympathetic ganglion. For occlusions of the upper limb, the stellate ganglion is employed; occlusions of the lower extremity indicate injection of the ipsilateral second lumbar ganglion. O'Leary[30] recommends the use of 20 cc. of 2 per cent procaine in producing the initial block. If so desired, a small polyethylene tube may be inserted through the needle and left indwelling for repeated blocks of 5 cc. of 2 per cent procaine every 2 hours. Massel[1] recommends the use of Bromsalizol as the agent of choice. The danger of hemorrhage into the retroperitoneal space when lumbar blocks are used in conjunction with anticoagulant therapy has been emphasized.[33]

Blocks should be performed as soon as the diagnosis of embolism is made.[32] If reflex vasospasm can be reversed before propagating thrombosis is extensive, the danger of subsequent thrombosis becomes reduced as stagnation is relieved by increased blood flow through collateral vessels. Whether or not surgery is contemplated as part of the definitive management, the judicious use of blocks in the early treatment of embolism permits a more favorable prognosis. If the initial results of blocks are favorable, they may be continued for several days by polyethylene catheter as prophylaxis against secondary or postoperative thrombosis.

When embolectomy is contemplated in occlusions of the lower extremity, the operator may choose to administer a spinal anesthesia to relieve arterial spasm and to provide surgical anesthesia for the operation.

Anticoagulants. Until the discovery and commercial production of heparin and, more recently, the coumarin derivatives, progressive arterial thrombosis posed one of the greatest hazards to the patient stricken with embolism. Extensive thrombosis pervading the mainstem and collateral vessels repeatedly frustrated both conservative and surgical measures to restore the circulation. Relief of vasospasm and embolectomy could offer little if secondary clotting occurred. Today, the early use of the appropriate anticoagulant, combined with other methods of management, restricts the appearance of this dangerous complication. However, once thrombosis has occurred,

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The site of Dramamine's action is probably in the labyrinthine structure.

*Swartout, R., III, and Gunther, K.: "Dizziness:" Vertigo and Syncope, GP *8*:35 (Nov.) 1953.

the prophylactic value of heparin or dicumarol is largely lost, though it has been shown experimentally that heparin given in the early stages of clot formation may effect a dissolution of the thrombus.[34]

Administration of anticoagulants should proceed as soon as the diagnosis of embolism is made. Heparin, by virtue of its prompt action, is the agent of choice in the initial treatment. Dicumarol, which does not produce therapeutic effects for about three days, may be given with the initial dose of heparin, but the latter must be continued until the prothrombin activity has fallen to a therapeutic level. Heparin may be administered intravenously in doses of 50 mg. every 4 hours[7, 27] until the clotting time of the blood is about 3 times normal or 15 to 30 minutes by the Lee-White method.

The duration of effect of a single intravenous dose is reported by Murray[35] to be about 90 minutes, though larger doses are effective for longer periods. By using depo-heparin, a single daily injection will maintain the clotting time at therapeutic levels, the dosage being adjusted by daily laboratory checks. De Takats, in studying the effects of sulfur compounds on the clotting mechanism, found that these agents increase the heparin response.[36] He suggests giving 0.6 gm. of sodium tetrathionate in 1,000 cc. of fluid by the intravenous route.

If one prefers to continue treatment with dicumarol, 300 mg. of this drug may be given orally on the first day, 200 mg. on the second day, and 100 mg. daily thereafter.[7] Heparin is discontinued on the fourth day of dicumarol administration. The dicumarol dosage is adjusted during therapy to maintain the prothrombin activity at about 20 to 35 per cent of the normal control value. Treatment with anticoagulants should be continued for one to three weeks.[7, 30]

Mechanical Devices to Improve the Circulation. Of controversial value in the management of peripheral embolism is the use of intermittent venous occlusion and alternating positive-negative pressure (pavex) devices. Both methods are designed to produce rhythmic changes in the venous pressure and are claimed by their respective advocates to increase the circulation of the ischemic limb.

The pavex apparatus consists of a boot which fits over the extremity with an air tight seal. The pressure within the boot is alternated between negative and positive by means of an electrically driven pump. Atlas[27] regards pavex as being of definite value and employs it for the relief of vasospasm. Dickinson[26] has found it useful in establishing the collateral circulation. Linton[37] claims that when used with sympathetic blocks, pavex increases the collateral flow and decreases the hazard of secondary thrombosis.

In 1937 Linton[22] compared the results of simple symptomatic treatment, pressure treatment, and embolectomy. He reported that 60 per cent of limbs treated with pavex were saved, whereas only 33 per cent survived when embolectomy was performed. This latter figure was little better than the 29 per cent salvage rate for limbs treated symptomatically.

Saland[18] recommends 100 hours of suction-pressure treatment. He states the contraindications to be pain, edema, or infection. The pain can often be diminished if high negative pressures are not employed. In spite of its many advocates, dissenting voices report that pavex is not effective in increasing blood flow.[21] Injudicious use of high negative pressures may actually traumatize the devitalized tissue of the ischemic limb.

Intermittent venous occlusion also has been considered as an effective means of increasing the peripheral circulation. Collens and Wilensky[38] suggest using a four-minute cycle, the tourniquet being inflated to 80 mm. Hg. for 2 minutes, then released for 2 minutes. They advise continuous use for 12 hours daily. Linton et al.[39] reported that inflation of the cuff to 100 mm. Hg. more than doubled the blood flow in the iliac artery. Cullen et al.[40] assert that no benefit accrues from venous occlusion. Other observers report that venous occlusion diminishes the venous oxygen saturation and interpret this as evidencing a decrease in circulation.[41]

The status of the above described procedures in the treatment of peripheral embolism is yet undecided. These mechanical methods are perhaps of greater use in the management of chronic arterial insufficiency. If one chooses to employ these techniques, they may be used either as part of the conservative regimen or as an adjuvant to embolectomy.

Surgical Procedures. With the development of anticoagulants and methods of relieving vasospasm, surgical treatments of embolism have produced increasingly favorable results. These procedures, which may be used singly or in combinations, include embolectomy, arterectomy, sympathectomies, vascular grafts, and amputations.

The procedure of embolectomy may be performed by a variety of techniques, all of which include the removal of the embolus through an arteriotomy wound near the site of occlusion. The aim of embolectomy is to restore blood flow through the occluded vessel by removing the obstruction. Performed in the early hours of occlusion, before thrombosis has involved the distal artery and collateral channels, embolectomy may effect a normal blood flow to the limb.

De Takats[23] lists the indications for embolectomy as (1) duration of occlusion no more than 10 hours, (2) maintenance of the general circulation, (3) lack of severe peripheral arteriosclerosis, and (4) failure to improve with conservative measures. As with all

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indications for therapy, these cannot be rigidly applied.

Andrus[42] would operate as late as 12 hours. While all authors agree that results are consistently better if surgery is performed within the first 10 hours, it is difficult to say at which point a patient should be denied embolectomy. Warren and Linton[2] state that surgery should not necessarily be abandoned after the "deadline"; they substantiate this view by the report of a successful femoral embolectomy performed 60 hours after occlusion. Olwin's group[43] reports cases operated upon as late as three months after occlusion. In such instances the collateral circulation has been adequate to provide a basal nutrition; removal of the primary obstruction reduces the degree of functional impairment due to persistent ischemia. Failure of the general circulation is not a strict contraindication to surgery, for the problem can often be controlled by digitalis and methods to relieve shock.

Though de Takats and others[30] recommend a trial of conservative management, other writers[4, 32] feel that the patient should be prepared for surgery as soon as the diagnosis is made. The justification for such an attitude is indicated by the frequent observations that though gangrene may not develop, there is residual functional impairment if the main arterial channel remains obstructed.[2, 42] This impairment may be of such severity as to make the patient a vascular cripple.

As with the previously mentioned contraindications, peripheral arteriosclerosis does not necessarily forbid embolectomy. Though arteriosclerosis is often considered a reason for failure of embolectomy,[32] careful technique may give the desired results. Since thrombosis is more likely to occur in a sclerotic artery, heparin should be given preoperatively. Irrigation of the arteriotomy wound with a heparin solution prior to closure is a further precaution against postoperative thrombosis.[4] Swinney and Coffen[44] state that even in elderly individuals the operator may usually find the arterial wall good enough for closure.

The site of occlusion also affects the decision to perform embolectomy. When the occlusion occurs in larger vessels, the technical difficulties of operation are minimal. In smaller vessels such as the radial, ulnar, tibials, and popliteal, closure of the arteriotomy wound may of itself produce obliteration of the artery. When embolism occurs in these smaller vessels, non-operative management is indicated. Due to the more extensive collateral circulation of the upper extremity, emboli to the arm rarely lead to gangrene. Therefore, a trial of conservative procedures may be more strongly considered than when occlusion occurs in the leg, though one must consider the possibility of impairment due to chronic arterial insufficiency.

Before embolectomy is performed, the patient should be prepared with blocks and heparin. Cardiac function should be restored in so far as possible. Local anesthesia is considered the least dangerous for severely ill patients,[2, 21] though spinal anesthesia is probably safe for procedures on the lower extremity.

Following routine preparation of the operative field, the tissues overlying the vessel are incised and the artery is opened by a longitudinal arteriotomy wound. Removal of the embolus and the distal thrombus is attempted by use of forceps, probes, and suction. Lavage of the vessel with a citrated solution may aid in the removal of small fragments of thrombus. Massive pulsating hemorrhage from the proximal artery and adequate reflux flow from the distal segment are the criteria for adequate removal of the clot and should be obtained before closure is made. Irrigation of the arterial lumen with a heparin solution aids in the prevention of postoperative thrombosis.[4] For cases in which extensive thrombosis has developed prior to surgery, Keeley and Rooney[45] describe the technique of retrograde milking as an adjunct to embolectomy. The thrombus is milked from the distal vessels toward the arteriotomy by wrapping the leg firmly from the toes upward with a wide para rubber bandage.

Embolectomy combined with sympathetic blocks, heparin, protection of the limb, and, possibly, vasodilatory drugs offers at present the most favorable prognosis for limbs with early embolism. Table V shows the limb salvage rates reported by several authors.

TABLE V

COMPARATIVE LIMB SALVAGE RATES FOR EMBOLECTOMY AND CONSERVATIVE METHODS

Author	*Embolectomy*	*Conservative*	*Comments*
Linton (1937)	33.3 %	60.0%	no blocks or anticoagulants
Warren and Linton (1948)	85.7 %	65.8%	
Reynolds and Jirka (1944)	81.25%		
Veal (1951)	81.2 %		

Arterectomy—the excision of the arterial segment occluded by embolism—may at times be used when embolectomy is contraindicated. McClure and Harkins[21] list the indications for arterectomy as (1) poor condition of the patient, (2) damage of the arterial wall, or (3) post-embolectomy thrombosis. The rationale of arterectomy is to bring about a cessation of the arterial spasm blocking the collateral vessels by resecting the site of endothelial irritation.[28] Since removal of the main arterial trunk places the burden of circulation upon the collateral vessels, the integrity of these channels must be determined before arterectomy is contemplated. De Takats[23] suggests that the procedure is contraindicated if sympathetic blocks preoperatively fail to improve the collateral flow. Leriche, Fontain, and Dupertuis[46] remark that several

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Although the nutrient needs for optimal health in the aged are not known to differ significantly from those in younger adults,[1] it has been shown that the daily protein requirements in elderly patients vary from person to person. Ascertained values range from below to above allowances recommended for persons in earlier years of adulthood.[2]

According to criteria such as "physical activity, gastrointestinal structure and function, pathologic disturbances, and chemical balances," it is suggested that an optimal diet for the elderly patient should provide at least 20 per cent of its calories in the form of protein.[3]

For several reasons this high intake of protein appears desirable. Decreased activity in the aged tends to induce loss of tissue protein. Preservation of protein enzymes and of endocrinal harmony necessary for supporting anabolic processes requires adequate protein nutrition. Also, the aged person usually is able to handle the end products of protein metabolism satisfactorily.

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inches of the occluded vessel must be resected to eliminate the reflex spasm. The first distal collateral, however, should be left intact.[21]

Vascular grafts: Though arterectomy may be useful in avoiding the immediate danger of gangrene, extremities so treated are susceptible to chronic arterial insufficiency. The function impairment related to this persistent ischemia may be avoided or alleviated if the continuity of the vessel is restored by vascular grafts.[47, 48] Blakemore, Lord, and Stefko[49] describe a non-suture technique of blood vessel anastomosis which is applicable to these cases. A vein segment is pulled through a vitallium tube and the ends are everted over the tube by 8 to 9 mm. The arterial ends are then slipped over the prosthesis and tied in place by ligatures.

Sympathectomies: The performance of lumbar sympathectomy upon a critically ill patient is seldom warranted. So prolonged and radical an operation should hardly be considered as part of the emergency management of embolism. It would seem more reasonable to reserve this procedure for the relief of subsequent arterial insufficiency.

Periarterial sympathectomy, on the other hand, is an innocuous procedure which may be performed as an adjunct to embolectomy. It serves the double purpose of removing the damaged adventitia and of preventing the conduction of sympathetic impulses to the vessel.[21] The effectiveness of this procedure has not been carefully evaluated.

Amputation will be the final treatment for a considerable number of limbs affected by embolism. In Haimovici's series,[3] 28 per cent of cases developed gangrene. Warren and Linton[2] reported that 30 per cent of limbs were lost. The time for amputation is when it is obvious that the limb cannot survive. However, even in serious cases where gangrene appears inevitable, a trial of surgical and medical efforts to save the limb should be attempted before amputation is considered. Removal of the limb is indicated when gangrene has persisted for 48 hours.[21]

Enzymatic Agents. In recent years the enzymatic digestion of intravascular thrombi has received the attention of several investigators. Though results of treatment in experimental animals have been observed, evaluation of these agents in the management of human embolism is yet inadequate. Innerfield's group,[50] working with experimental animals, evaluated the anticoagulant, fibrinolytic, and thrombolytic effects of intravenous trypsin. They reported prolonged clotting time and diminished titers of the protein constituents of the clotting mechanism. Higher dosages of trypsin would effect dissolution of thrombi. This same group of investigators,[51] working with humans, found trypsin to be a safe and effective means of destroying the clotting mechanism. Untoward results were not seen with careful calculation of dosage.

The suggested method of preparation is to dissolve 10 mg. of trypsin in 3 cc. normal saline and then add to 100 cc. of normal saline containing 1 cc. of Histadyl. The solution is given intravenously at the rate of 30 drops per minute. The infusion may be repeated twice daily. The prepared solution must be given promptly as 40 to 50 per cent of its proteolytic activity is lost within 3 to 4 hours.

Sherry et al.[52] studied the effects of trypsin, chymotrypsin, and plasmin activator on one-day-old femoral and popliteal thrombi in dogs. Their studies reveal no clot dissolution by trypsin, but chymotrypsin in doses of 12 to 30 mg./Kg. accomplished complete lysis of the clots in half the animals studied. The most striking effects were noted in a group of animals injected with a mixture of streptokinase and a partially purified human plasma fraction III factor. Extensive lytic effects on the thrombi were noted in the majority of animals in this latter group. Further clinical investigation is necessary before the enzymatic agents will become a part of the routine management of peripheral arterial embolism.

PROGNOSIS

Prognosis of the Limb. The prognosis of a limb affected by arterial embolism is determined largely by the degree and duration of ischemia. The several variables which influence ischemia have been explained to some extent, but many aspects of the problem are not yet elucidated. The ischemia—hence the survival of the limb—depends upon the site of occlusion, the severity and duration of spasm, the extent of secondary thrombosis, the presence of prior vascular pathology, the presence of collateral vessels, the general circulatory reserve, and the promptness of treatment. Haimovici[3] notes that the age of the patient is not an important factor.

Though one can describe the extent of vasospasm and thrombosis as influencing the prognosis, it is more difficult to explain the case to case variation of these factors. Why does one patient with a femoral embolus develop a line of demarcation near the groin, while another with less extensive vasospasm will form a level of ischemia near the knee? Why does one patient within 10 hours have extensive obliteration of his vessels by secondary thrombosis, though another, even without treatment, develops minimal thrombosis? Haimovici[3] has recognized this case to case variation and describes the following clinical forms of embolism:

1. Embolism with marked ischemia and early death —11.3 per cent of cases

2. Embolism with marked initial ischemia which partially subsides, resulting in chronic post-embolic ischemia—16.5 per cent of cases

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3. Embolism followed by gangrene—28 per cent of cases

4. Embolism with marked initial ischemia which subsequently subsides completely, resulting in anischemic embolism—29.5 per cent of cases

5. Embolism with undetermined degree of recovery due to inadequate follow up—9.6 per cent of cases

6. Silent embolism—5.7 per cent of cases.

With such marked variation in the natural course of the disease, the difficulties in assessing the various therapeutic methods are immediately apparent.

In discussing prognosis of the limb it is noted that there are three groups or classifications of results of embolism: (1) limbs with no residual ischemia, (2) limbs with some degree of residual ischemia, and (3) limbs developing gangrene and requiring amputation. Most authors, in assessing their results, consider only whether the limb was saved, making no analysis of the frequency of residual ischemia. With adequate early treatment, 60 to 85 per cent of limbs may be saved.[2, 32] Reynolds and Jirka[7] state that they obtained functional limbs in 81.25 per cent of cases. According to Haimovici,[3] 16.5 per cent of cases will develop chronic post-embolic ischemia manifested by Volkmann's contractures, intermittent claudication, ischemic neuritis, persistent color changes, and small patches of gangrene. Griffiths[53] reported that 3 of 20 cases developed typical Volkmann's ischemic contractures despite apparently successful restoration of the circulation.

Prognosis of the Patient. In cases of peripheral arterial embolism, the ultimate prognosis of the patient is that of the causative disease.[54] Haimovici[3] reported his case mortality rate to be 47.6 per cent in untreated and conservatively treated cases and 51.7 per cent in those treated surgically. Pratt[4] remarks that the survival charts of embolism closely resemble those for well-treated carcinoma of the breast. Whether embolism occurs in the upper or lower extremity apparently does not affect the mortality rate.[55] The hospital mortality reported by other authors is 32 to 38.7 per cent.[2, 42] Causes of death are usually on the basis of the primary heart disease or due to visceral emboli. The concurrent venous thrombosis of peripheral embolism not infrequently complicates the picture by producing pulmonary embolism.

PROPHYLAXIS

With modern therapeutics placing increased emphasis on preventive medicine, the application of this philosophy to arterial embolism should be considered. A perspective of the problem may be gained through the statistics of Pratt,[4] who reports that embolism occurs in 25 per cent of patients dying of heart disease. The problem arises in about 10 per cent of patients with rheumatic heart disease, and about 60 per cent of these will have multiple episodes.[9] The incidence of thrombo-embolic episodes following myocardial infarction ranges from about 15 to 25 per cent.[14, 15, 16]

The methods of attack upon the problem are both medical and surgical. Long term anticoagulant therapy has been employed by Cosgriff[9] in recurrent embolism of cardiac origin. In a series of patients who had had 103 embolic episodes during 275 "patient-months," long term therapy with dicumarol reduced the frequency of embolism in these same patients to 13 episodes during 625 "patient-treatment months." Foley and Wright[56] reported that no thromboembolic episodes occurred in their patients while the prothrombin time was maintained at therapeutic levels.

The frequency with which auricular fibrillation is associated with embolism has led to efforts to convert this arrhythmia as a prophylactic measure. Harvey and Levine[12] report that 90 per cent of persistent fibrillators develop auricular thrombosis and, thus, are potential embolizers. The conversion of fibrillation to a regular rhythm is attempted by administering appropriate doses of quinidine or pronestyl. Though some authors feel that conversion itself may dislodge thrombotic fragments into the circulation,[57] Lackay and Housel[58] regard this danger as no greater than if fibrillation should continue.

Deserving only brief remark is the application of surgery to the prevention of embolism. The procedure of left auricular appendectomy has not met with great acclaim; it would seem to be an unreasonably dangerous procedure in patients whose chances for survival are poor. Nor could the procedure logically be effective, for many cases do not have the thrombotic process restricted to the appendage. Leonard and Cogan,[59] reporting 8 cases, noted that 3 re-embolised in a matter of days. They remark that 25 per cent of embolic sources are restricted to the appendage of the left auricle; it is impossible to determine this 25 per cent of patients pre-operatively. Jordan et al.[60] analyzing 57 cases of mitral stenosis with cardiac thrombi, found that about half of the thrombi on the left side were not restricted to the appendage. When the source of potential emboli is an aneurysm of the aorta or a major artery, the surgeon may resect the aneurysm and substitute a vascular graft.[48]

CASE REPORT

Patient C. S., a 53-year-old colored man, was admitted on the evening of May 5, 1953, to the Kansas City Veterans Administration Hospital with a complaint of severe pain in the right leg. He noted nothing unusual referable to the extremity until about 7 days prior to admission when, on arising one morning, he noticed mild swelling, subjective coldness, and slight aching in the right foot. He spent the next several days quietly at home and observed that the

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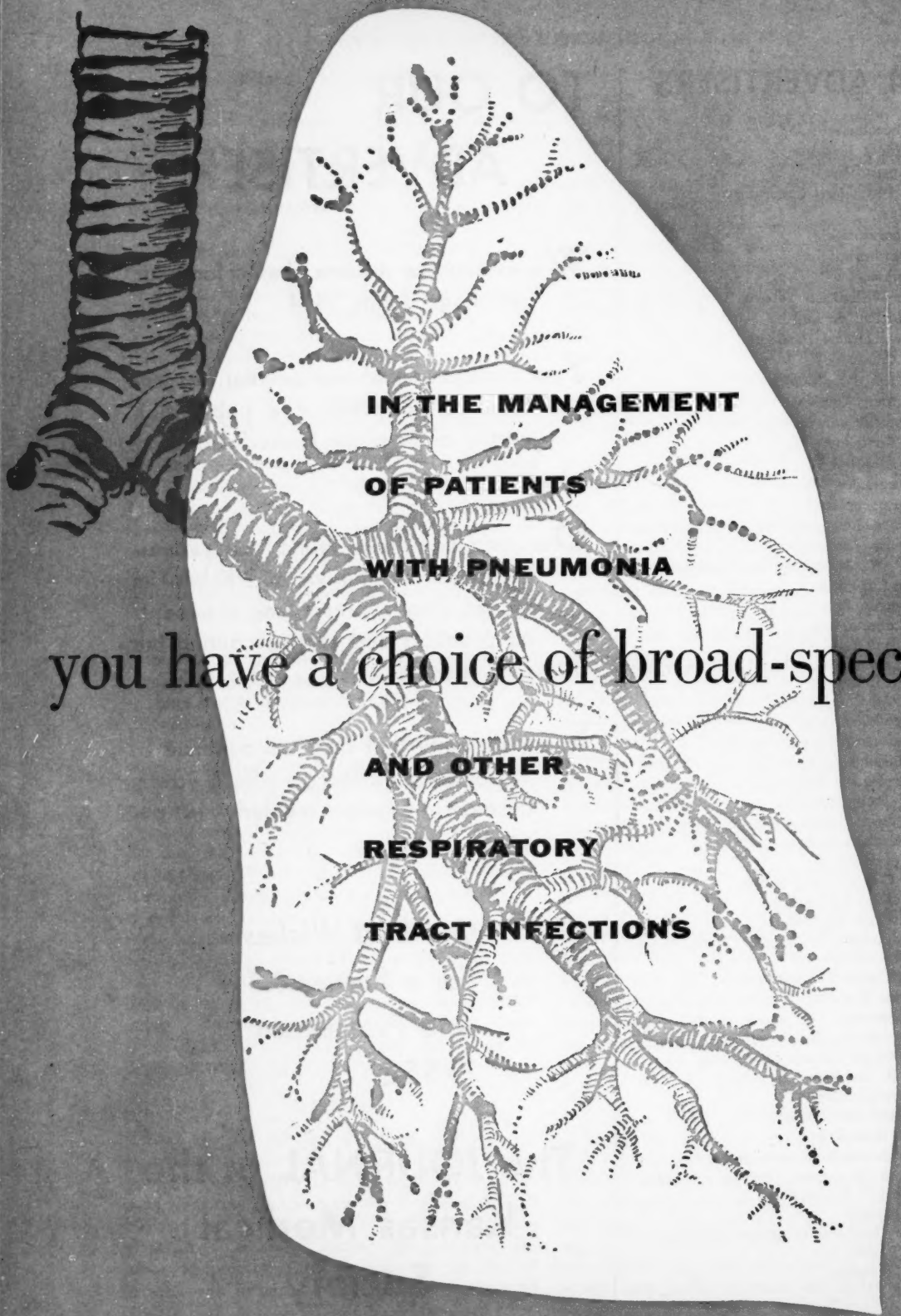

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1. *O'Regan, C., and Schwarzer, S.: J. Pediat. 44:172 (Feb.) 1954.*
2. *Waddington, W. S.; Bergy, G. G.; Nielsen, R. L., and Kirby, W. M. M.: Am. J. M. Sc. 228:164 (Aug.) 1954.*

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aching and swelling disappeared though the coldness persisted.

No further difficulty was experienced with the leg until about 3:00 p.m. on the afternoon of admission. The patient had been resting quietly in a chair when the phone rang; as he arose to answer, the right knee seemed to give way beneath him and the leg was very weak. He returned to his chair and shortly noticed a mild aching which became progressively worse until it was severe. He called his physician who gave an injection for pain. He was brought to the hospital and became comatose during the trip.

The patient later stated that he had not felt well since 1936 and that during the past 10 years he had suffered recurrent episodes of vertigo. In 1949 he consulted a physician for symptoms of frequent severe temporal headaches, vertigo, diplopia, faintness, and dyspnea; he was given digitalis. In 1950 he entered a hospital because of similar symptoms; he was told at this time that he had high blood pressure and an enlarged heart. In 1951 he was admitted to another hospital complaining of severe pain in the left leg. A diagnosis of femoral embolism was made, and the limb was amputated in the proximal third of the thigh.

The patient had taken digitalis erratically since 1949, but because of financial problems he had taken none in the past 6 months. During this period he had developed progressively severe symptoms of cardiac decompensation—exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. All of these symptoms had become dramatically worse during the past week.

Past History: The patient had measles, mumps, and pertussis during childhood without complications. He had typhoid fever in 1909 and gonorrhea in 1920. He underwent an appendectomy in 1950 and amputation of his left leg in 1951. He was employed as a jailer until May, 1951, when he was forced to stop work because of his cardio-vascular status.

Review of Systems: The patient's general health had been poor since 1936. During the past 6 to 7 years he had had frequent headaches, vertigo, diplopia, and faintness. During recent years he had suffered frequent respiratory infections and a chronic cough productive of small amounts of blood tinged sputum. His appetite had been poor since 1952, and he experienced frequent nausea but no vomiting. His stools were occasionally black and tarry, but more often were chalky white. He noted nocturia about 4 times nightly.

Physical Examination: Examination revealed a well developed, well nourished colored man who was comatose and in acute respiratory distress. The head and neck revealed no scars; the thyroid was smooth and not enlarged; the trachea was in the mid-line. The pupils were found to be small and equal, and they reacted to light; the sclera and conjunctiva were normal. Funduscopic examination revealed gr. iii hypertensive retinopathy. The tympanic membranes and auditory canals were normal. The nasal cavities revealed no ulceration, septal deviation, or obstruction. Oral hygiene was poor. The breath was foul with a faint, sweetish odor superimposed. The tongue was red and moist.

Movements and configuration of the chest were bilaterally symmetrical. Respirations were irregular (Cheyne-Stokes) with periods of apnea lasting up to one minute. Tactile fremitus was normal, and there was no dullness to percussion. Moist rales were heard bilaterally in the lung bases. The left border of the heart was percussed in the anterior axillary line. The heart sounds were feeble, the rate 120; there was a fetal rhythm. The second pulmonic sound was accentuated.

The abdomen revealed no areas of rigidity, but shifting dullness was found in the flanks and a fluid wave was elicited. A blunted liver edge was palpated 6 centimeters below the right costal margin, and there was tenderness to fist percussion over the right lower thorax. The left leg had been amputated at the proximal third of the thigh. On the right the femoral, popliteal, dorsalis pedis, and posterior tibial pulses were absent. The extremity was cold, the nail beds blanched, and a line of ischemia was found immediately distal to the inguinal ligament. There was marked muscular weakness and a loss of deep reflexes in the limb. A sensory examination was impossible because of the patient's inability to respond.

The initial impression was:

1. Arterial embolism of the right common femoral artery
2. Hypertensive cardiovascular disease with
   a. cardiac hypertrophy
   b. cardiac decompensation
   c. cardiac arrhythmia—fetal rhythm
3. Chronic passive congestion of the lungs and abdominal viscera.

The admission urinalysis report included specific gravity 1.017; albumin 4 plus; sugar negative; microscopic of 12 white blood count, six to eight granular casts, and one to two hyaline casts per high power field. The red blood count was 5,200,000; hemoglobin 14.5 gm./100 cc., white blood count 8,850 with a normal differential. Further blood studies revealed the following: nonprotein nitrogen 49.5 mg./100 cc., blood glucose 250 mg./100 cc.

Shortly after admission the patient was started on oxygen continuously by nasal catheter; rapid digitalization was carried out with two 0.6 mg. doses of digitoxin intravenously; 10 cc. of aminophylline were given slowly by vein. A spinal anesthetic was ad-

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ministered and continued at frequent intervals by means of an indwelling spinal catheter.

Under this management the patient rapidly regained consciousness and became well oriented. The Cheyne-Stokes type of respiration ceased, and the rhythm of the heart became normal with a rate of 100 to 120. The femoral pulsations returned, and the level of ischemia receded from the inguinal region to a point in the distal third of the thigh.

When it was felt that the patient's cardiac condition was adequately controlled, he was taken to the operating room where a common femoral embolectomy was performed under continuous spinal anesthesia. The clot was removed by means of a glass suction tip approximately 10 hours after the initial occlusion. A small amount of distal thrombus was obtained. A free flow of blood was obtained both from above and from below the arteriotomy wound. The artery was closed and the vessel irrigated with a heparin solution.

Immediately after the operation the popliteal pulse was faintly palpable, but the dorsalis pedis and posterior tibial arteries could not be felt. The line of ischemia had now receded to the knee. Postoperatively the patient was continued on depo-heparin, and the clotting time was checked daily.

On the second hospital day the calf of the right leg was markedly swollen and tender. The popliteal and femoral pulses were strong, but the ankle pulses were still absent. The line of ischemia was now in the mid-calf region. Cardiac decompensation manifested by Cheyne-Stokes breathing, fetal rhythm, and increasing pulmonary edema and ascites again appeared, but was quickly controlled with digitalis.

On the fourth hospital day the clotting time was more than one hour, and on the fifth day the patient began to bleed from the operative wound. On the sixth day the patient was returned to surgery where the blood clot was evacuated from the wound and the opening closed secondarily. By the seventh day the congestive failure was well controlled; the ankle pulses were yet absent, but there was good color return to the nail beds; a sensory deficit persisted in the lower part of the leg. The patient continued to improve and was discharged to the old soldiers' home approximately two months after the embolic accident. The final diagnosis was femoral embolism with residual chronic ischemia.

COMMENT AND SUMMARY

Peripheral arterial embolism usually appears as a serious complication of grave cardiac disease. Rheumatic heart disease and myocardial infarction are the most frequent causes of embolism. Emboli may lodge in any artery of the body, but 60 per cent or more occlude vessels of the extremities, the incidence being three to four times higher in the legs. Acute occlu-

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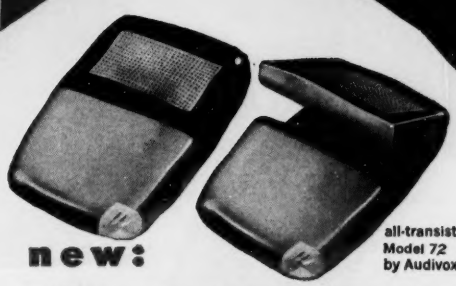

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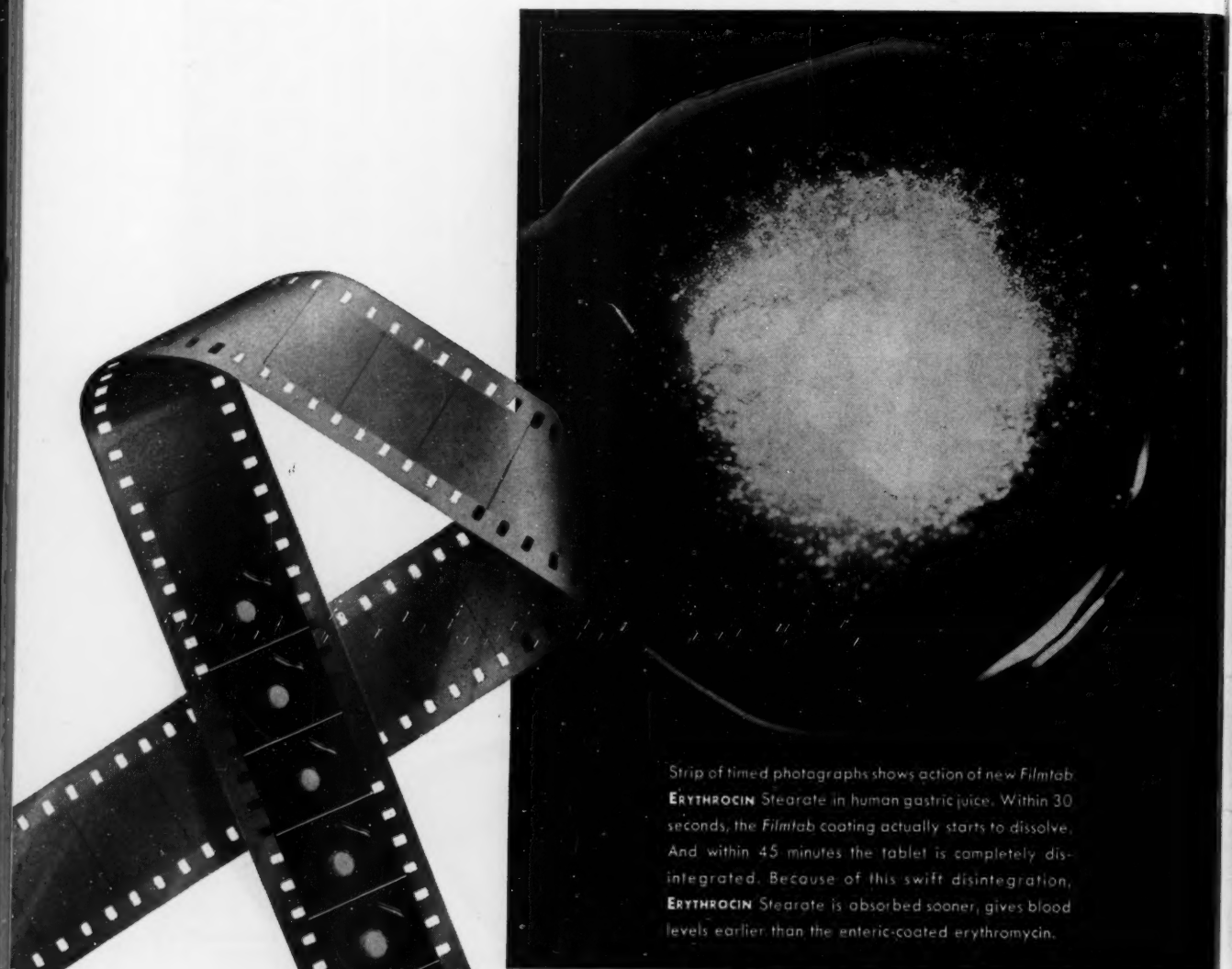

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sion initiates a series of events which contribute further to the ischemia, namely arterial spasm, secondary thrombosis, arteritis, and thrombophlebitis.

The treatment of embolism includes protection of the limb, vasodilator drugs, anticoagulants, mechanical pressure devices, surgical procedures, and enzymatic agents.

The prognosis of the limb affected by embolism depends upon the extent and duration of ischemia. Approximately 80 per cent of limbs may be saved with early adequate treatment, though some of the surviving limbs will develop chronic ischemia. Many of the patients die during the hospital stay, the case mortality rate being in the range of 30 to 50 per cent.

Certain prophylactic procedures have been attempted to prevent recurrent embolism. These include anticoagulant therapy, conversion of auricular fibrillation, left auricular appendectomy, and resection of arterial aneurysms.

A case is presented which demonstrates the following characteristics of the syndrome of acute arterial embolic occlusion:

1. the precarious condition of the patient
2. the recent change in cardiac status
3. the tendency for the occurrence of multiple embolic episodes—two femoral emboli
4. the lack of initial severe pain
5. the effect of vasospasm and the relief of spasm by spinal anesthesia
6. the safety of spinal anesthesia when the cardiac reserve is poor
7. the favorable results of early application of combined medical and surgical measures
8. the development of chronic arterial insufficiency.

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THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

Federal Aid

This nation's 80 medical schools are graduating 6,700 new physicians a year. That is far too few to replace the doctors dying and retiring each year, and to meet the nation's growing medical demands.

State taxation and private donations have been stretched about as far as they will go to improve the situation by building bigger and better medical schools. They won't stretch far enough.

A solution which more and more medical educators are eyeing is federal support. The schools and their supporters are reluctant to accept such a solution, and rightly so. We don't want congressional interference in the operation of our medical schools. We don't want political medicine.

But we have this ridiculous paradox where federal funds are used for the cure and prevention of hog cholera in Iowa and the control of locusts in Trans-Jordan, but we're afraid to use those same funds to improve medicine in this country.

It can be done—federal funds can be used safely for the support of medical schools. Legislation could be drawn which specifies clearly that the federal government has no concern with the hiring or firing of faculty, or the establishment of curriculum. Any school would be eligible for aid which meets the standards of the American Medical Association and those standards would be established by the Association with no interference by the federal government.

Aid could take two forms. Outright grants could be made to schools for construction and equipping of classrooms, laboratories and hospitals. Federal funds could be used for scholarships for competent students under an arrangement similar to the GI bill for education of veterans. The medical school's faculty, and not the government, would determine the student's eligibility for the aid.

It is wise to be skeptical of federal aid and alert to its dangers. But fear shouldn't be permitted to block the wisest use of the nation's resources in meeting the nation's needs.—*Chanute Tribune, November 27, 1954.*

Naps Instead of Pills

Chosen as the "Family Doctor of 1954," Dr. Karl B. Pace of Greenville, N. C., shows his appreciation of the honor by giving much-needed advice to this generation suffering from nerve tension, dignified by such alarming phrases as psychosomatic and emotional troubles, affecting at least 60 per cent of the population. He has set a sort of free clinic for those who are finding the going a bit tough in an age of stress and strain.

His formula for happy and healthy living is simple enough. Live today, let tomorrow take care of itself. Don't try to get rich. Try to like everyone you meet, spend more time at home, and take a nap in the middle of the day as a means of relaxation. The family doctor today must be a counselor rather than a physician. Give advice instead of pills.

Sound advice, every word of it. But the average doctor will tell you he wouldn't get very far, or be too popular, by prescribing naps instead of medicine. As a people we are looking for something to take the place of common sense. We are so busy trying to keep up with the procession we do not have time for midday naps. How much better off we would be if we followed the advice of the good doctor. But we won't because it is free, and there are no prescriptions to be filled.—*Wichita Eagle, December 1, 1954.*

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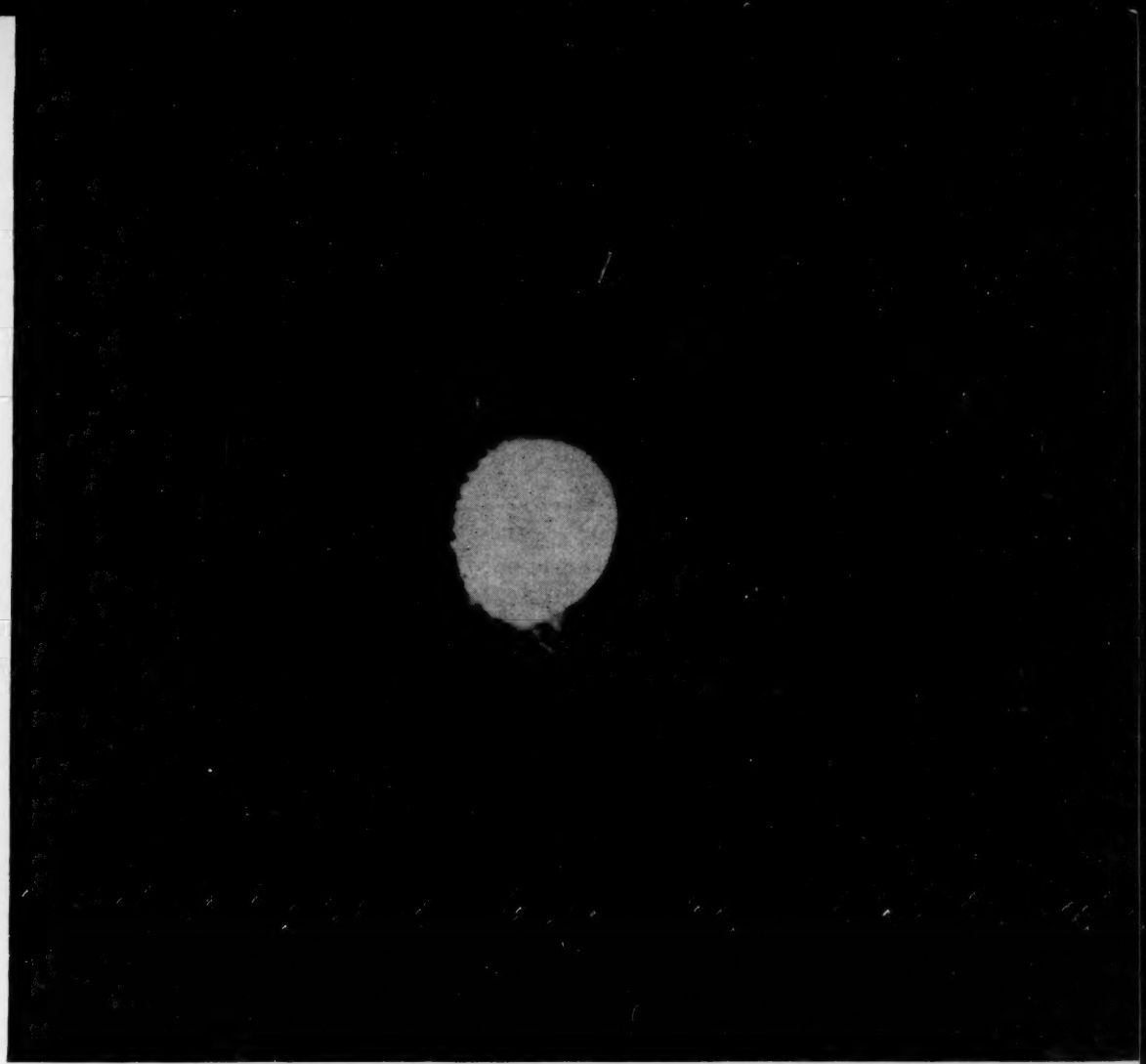

ELECTRON PHOTOMICROGRAPH

Staphylococcus aureus 44,000 X

Staphylococcus aureus (Micrococcus pyogenes var. aureus) is a Gram-positive organism commonly involved in a great variety of pathologic conditions, including

pyoderma • abscesses • empyema • otitis • sinusitis • septicemia

bronchopneumonia • bronchiectasis • tracheobronchitis • and food poisoning.

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ANNOUNCEMENTS

Announcement is made of a heart conference to be presented February 10 and 11 by the University of Kansas School of Medicine in co-operation with The Kansas Medical Society, the Kansas State Board of Health, the Kansas Heart Association, and the Kansas City (Missouri) Heart Association. There will be no registration fee.

The 7th annual meeting of the American Academy of Forensic Sciences will be held in the Biltmore Hotel, Los Angeles, February 17, 18, 19. The Law Department of the A.M.A. has urged that the profession take an increasing interest in medico-legal problems. Further information may be obtained from Dr. Frederick D. Newbarr, 109 Hall of Justice, Los Angeles 12, California.

The 9th annual M. D. Anderson Symposium on Fundamental Cancer Research will be held March 10, 11, and 12 at the University of Texas M. D. Anderson Hospital and Tumor Institute in the Texas Medical Center, Houston. The first day will be devoted to a review of current research projects at the hospital, the second day to histochemistry, and the third day to presentations reflecting current cancer research in the Southwest.

The Ciba Foundation, London, a body for the promotion of international cooperation in medical and chemical research, announces that it will present five awards of 300 pounds each to contestants doing experimental research into problems of aging. Entries, to be submitted no later than February 28, will be judged by an independent international panel of scientists. Director of the foundation is G. E. W. Wolstenholme, 41, Portland Place, London, W.1, England.

The Harvard University School of Public Health announces that candidates for scholarships in public health must present applications by March 1, 1955. Awards range from part tuition to tuition plus a stipend, according to the qualifications and financial needs of applicants. Complete information may be secured from the secretary, Harvard School of Public Health, 55 Shattuck Street, Boston 15, Massachusetts.

The 18th annual meeting of the New Orleans Graduate Medical Assembly will be held March 7-10, 1955, with headquarters at the New Orleans Municipal Auditorium. The post-clinical tour to Europe will follow immediately after the meeting. Complete information may be secured from Dr. Maurice E. St. Martin, 1430 Tulane Avenue, New Orleans 12, Louisiana.

The Council on Postgraduate Medical Education of the American College of Chest Physicians, in cooperation with the staffs and faculties of the hospitals and medical schools of Philadelphia, will sponsor the 8th annual Postgraduate Course on Diseases of the Chest at the Bellevue-Stratford Hotel, Philadelphia, March 7-11. Tuition is $75. Information may be secured from the College, 112 East Chestnut Street, Chicago 11, Illinois.

A five-day postgraduate course on "The Clinical Management of Emotional Problems in Children" will be presented at the University of Colorado Medical Center, April 4-9. Registration will be limited. The guest lecturer will be Dr. Reginald S. Lourie, director of the Psychiatric Clinic of Children's Hospital, Washington, D.C. Program and information may be obtained from the Office of Postgraduate Medical Education, 4200 East Ninth Avenue, Denver 20, Colorado.

The Council on Undergraduate Medical Education of the American College of Chest Physicians offers three cash awards for the best contributions prepared by undergraduate medical students on any phase of the diagnosis and treatment of heart or lung disease. Awards will be $250, $100, and $50. Closing date of the contest is April 10. The address of the College is 112 East Chestnut Street, Chicago 11, Illinois.

The Mayo Clinic and Mayo Foundation announce a four-day program, April 19-22, of lectures and discussions on problems in the general fields of medicine and surgery. Attendance will be limited, so physicians wishing to attend should communicate with Dr. N. W. Barker, Mayo Clinic, Rochester, Minnesota, before March 1. No registration fee will be charged.

The U. S. Civil Service Commission has announced that applications are now being accepted for medical officer for trainee positions in St. Elizabeth's Hospital, Washington, D.C. Rotating interns will receive

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$2,800 a year, and residents-in-training in psychiatry and in neurology will receive $3,400 to $4,200 a year. Appointments will be open July 1, but applications will be accepted at any time. Information may be secured from post offices or from the U. S. Civil Service Commission, Washington 25, D.C.

BOOK REVIEWS

Clinical Aspects of the Autonomic Nervous System. By L. A. Gillian. Published by Little, Brown and Company, Boston. 316 pages. Price $6.50.

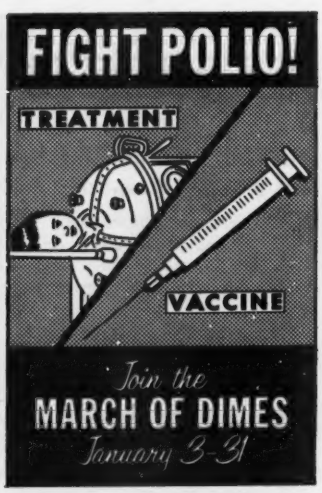
Fundamentally an anatomy test, this small volume presents a detailed account of the autonomic nervous system in a fashion pleasing and helpful to the clinician. It is up to date with the most recent contributions in neuroanatomical and neurophysiological research, and it is modern in its explanation of many psychosomatic symptoms on the basis of autonomic dysfunction.

Its first half is a discussion of the anatomy of the autonomic nervous system as a whole, and its second half is organized into a presentation of the innervation of the individual organs and systems, with many comments of clinical significance.

The figures of this complicated subject are reasonably clear, and some are conveniently duplicated several times in the text. Chapters are outlined at their beginning, and section headings make for ready reference. An excellent bibliography is included.

The book as a whole is well done and will be of value to both anatomist and clinician.—*W.P.W.*

Total registration at the A.M.A. meeting in Miami last month was 7,707. Of this number, 3,253 were physicians.



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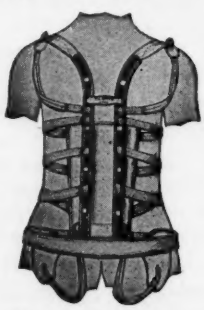

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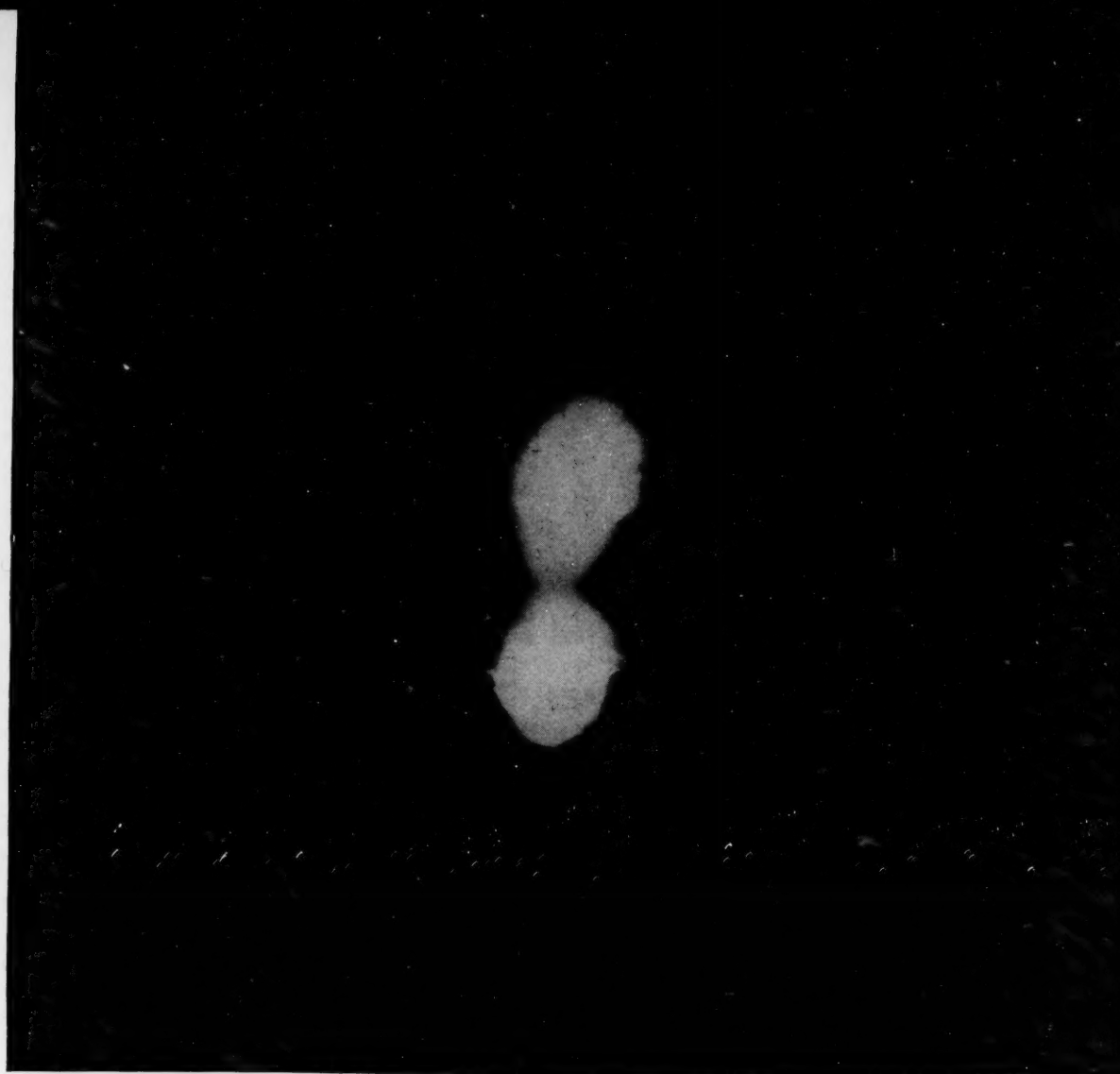

ELECTRON PHOTOMICROGRAPH

Streptococcus faecalis 40,000 X

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Index to Advertisers

Abbott Laboratories ..... 50 and 51
American Meat Institute 39
Ames Company, Inc. ..... Inside back cover
Audivox, Inc. ..... 48 and 49
Ayerst Laboratories XIII
Bayer Company 37
Brown and Williamson Tobacco Corporation IX
Chicago Medical Society XII
Ciba Pharmaceutical Products 47
Fairmount Hospital 60
Fazio Laboratories, Inc. 53
Goetze Niemer Company 53
Hanicke, P. W., Manufacturing Company 58
Kansas Oxygen, Inc. XVI
Lakeside Laboratories, Inc. V
Lattimore-Fink Laboratories 53
Lederle Laboratories Division, American Cyanamid Company ..... 22 and 23
Lilly, Eli, and Company XVIII
Lorillard, P., Company XIV
Mead Johnson and Company ..... Back cover
Medical Protective Company 53
Menninger Foundation 57
Merchants Finance Corporation, Inc. XVII
Munns Medical Supply Company, Inc. XVII
Mutual Distributors, Inc. .....
.....58
.....53
...over and III
.....57
d
.44 and 45
.....60
.....57
.....VIII
.....35
.....58
.....VI
e 53
.....XVI
55, and 59
.....XVII
.....X
.....XII

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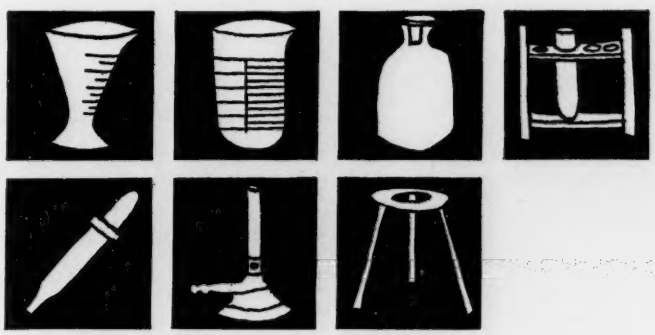

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